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# 31. The cat with signs of chronic vomiting

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## KEY SIGNS

- Active elimination of stomach contents through the mouth.
- $\pm$  Preceded by nausea, pacing, and salivation.
- Usually digested food or liquid with acidic or neutral pH.
- $\geq 2$  weeks duration.

## MECHANISM?

- Vomiting occurs when the vomiting center in the medulla or when afferent impulses from the cerebral cortex, chemoreceptor trigger zone (CRTZ), or receptors in the pharynx or abdominal viscera is stimulated.
- Vomiting is an **active process** that must be distinguished from regurgitation.
- Vomitus may consist of undigested food material (if swallowed whole), partly digested or even liquid, and may be clear, yellow (bile stained) or brown (food colored).
- The **pH of vomitus is usually acidic** ( $\text{pH} < 4$ ), but may be neutral ( $\text{pH} 7$ ) if duodenal content is present (bile reflux).

## WHERE?

- Chronic vomiting can be caused by **primary gastrointestinal diseases** or by **extra-intestinal diseases** or disorders that may have no apparent association with the GI tract.
- Vomiting is **rarely associated with primary colonic disease**.
- Extra-intestinal causes of vomiting include:
  - Hepatobiliary disease.
  - Pancreatic disease (e.g. pancreatitis, neoplasia).
  - Renal disease causing acute or chronic renal failure.
  - CNS disease (e.g. vestibular disease, encephalitis, seizure disorders, neoplasia).
  - Metabolic diseases (e.g. hyperthyroidism, ketoacidotic diabetes mellitus or Addison's disease).
  - Cardiomyopathy or congestive heart failure.
  - Systemic illness or infection affecting the CRTZ or cortex (e.g. septicemia).

## WHAT?

- Primary gastrointestinal diseases that cause chronic vomiting include:
  - Parasites (e.g. *Physaloptera*).
  - Infectious diseases (e.g. *Helicobacter*).
  - Inflammatory diseases (e.g. IBD, gastritis or gastric ulcer disease).
  - Neoplasia.

- Mechanical (e.g. antral pyloric hypertrophy/stenosis, obstruction, intussusception).
- Dietary disturbances (e.g. food intolerance or food allergy).

The **most common causes of chronic vomiting** are: **inflammatory diseases** (e.g. food allergy or IBD), **dietary** (dietary indiscretion or food intolerance), **neoplastic** diseases and **metabolic or extra-intestinal disturbances** (e.g. renal, hepatic or pancreatic disease).

## QUICK REFERENCE SUMMARY

### Diseases causing signs of chronic vomiting

#### ANOMALY

- Dysautonomia (p 695)

This is a disorder of the autonomic nervous system resulting in GI signs due to motility disturbances in the esophagus, stomach, small and large intestine. The esophagus and colon are most dramatically affected and thus regurgitation and constipation are more prevalent than other signs. Chronic vomiting may occur or regurgitation may be reported as vomiting. Acute onset of anorexia, depression, dilated pupils without blindness, prolapsed third eyelids and bradycardia are common.

#### MECHANICAL

- **Foreign bodies\* (p 688)**

Foreign bodies are often associated with an acute onset of vomiting, but string ingestion may cause chronic intermittent vomiting when the obstruction is incomplete or motility disturbances are intermittent.

- Antral pyloric hypertrophy/stenosis (p 694)

Acquired or congenital thickening of the pyloric tissue may result in vomiting due to motility disturbances, obstruction of outflow or both. Vomiting usually occurs several hours after eating and may be projectile. Rarely stenosis is functional with no evidence of thickening.

- Intussusception (p 692)

Intussusception is usually associated with acute onset of severe vomiting, but sliding, intermittent or incomplete intussusception may have chronic, intermittent vomiting.

#### METABOLIC

- **Hyperthyroidism\*\* (p 670)**

Intermittent vomiting occurs in conjunction with weight loss, polyphagia and polyuria. Other signs include hyperactivity, irritability, poor grooming habits and large, bulky or mucoid feces.

- **Hepatic diseases (hepatic lipidosis, cholangitis, hepatic cirrhosis, portosystemic anomalies, etc.)\*\* (p 674)**

Vomiting is often associated with hepatic diseases or failure (of any cause). Other signs of hepatic disease include fever, weight loss, icterus, anorexia and lethargy. Cats in severe hepatic failure may show signs of hepatoencephalopathy, including drooling, apparent blindness, stupor, pica or altered behavior.

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- **Chronic renal failure\* (p 680)**

Intermittent vomiting is due to the effects of azotemia and hypergastrinemia on the gastric mucosa. The most common signs are polyuria, polydipsia, weight loss and decreased appetite.

- **Congestive heart failure\* (p 689)**

Intermittent vomiting often occurs in the late stages of the disease, in addition to the signs of cardiovascular and respiratory distress or failure.

- **Diabetes mellitus\* (p 681)**

One third of cats with diabetes have a history of vomiting. Cats with severe ketoacidosis secondary to unregulated diabetes mellitus often present with signs of anorexia, weight loss and a sudden onset of vomiting and depression. Polyuria and polydipsia are also present and may have been ongoing for several months prior to the development of the severe signs of illness.

- **Hypercalcemia\* (p 683)**

Vomiting often occurs secondary to hypercalcemia. Other clinical signs associated with hypercalcemia include polyuria/polydipsia, anorexia and lethargy. Severe hypercalcemia may cause muscle weakness and dystrophic calcification of tissues (e.g. skin, kidneys, stomach, etc.).

- **CNS diseases (encephalitis, seizure disorders, vestibular disease, neoplasia)\* (p 690)**

Vomiting is an uncommon sign of neurologic disease, but chronic intermittent vomiting may be associated with many different diseases of the CNS, and should be considered whenever signs of neurologic disease coexist with vomiting, especially vestibular signs (e.g. head tilt, nystagmus, rolling) or signs suggesting hepatoencephalopathy (e.g. drooling, behavior changes or apparent blindness).

- **Hypoadrenocorticism (Addison's disease) (p 691)**

This is an uncommon endocrine disease in cats that often presents with only vague clinical signs of lethargy, anorexia and weight loss. Vomiting and diarrhea are less common signs in cats, as are changes in the electrocardiogram associated with hyperkalemia.

## NEOPLASTIC

- **Intestinal adenocarcinoma\*\* (p 677)**

Anorexia, weight loss and lethargy are the most common signs, but vomiting often occurs when the tumor reaches a size that causes motility disturbances or bowel obstruction. The most common location is the distal small intestine.

- **GI lymphosarcoma\*\* (p 675)**

This tumor tends to be more infiltrative and may involve large segments of small bowel, stomach or colon. Signs range from anorexia and weight loss to vomiting or diarrhea, depending on the location of the tumor.

- **Intestinal mast cell tumor\* (p 687)**

These tumors are often solitary and primarily affect the distal small intestine, and may cause no signs, other than weight loss and anorexia, until late in the disease when vomiting develops either secondary to tumor size or presence of metastatic foci.

- **Systemic neoplasia (generalized lymphoma, systemic mast cell tumor)\* (p 689)**

Vomiting may occur due to neoplasia involving abdominal organs resulting in direct effects, organ dysfunction or the release of vasoactive substances (e.g. mast cell tumor) that cause gastritis. The most common clinical signs associated with neoplasia include anorexia, weight loss or lethargy and usually occur in older cats.

- Other intestinal neoplasia (fibrosarcoma, carcinoids, plasmacytoma, leiomyosarcoma, etc.) (p 693)

The presence of vomiting as a clinical sign depends on the location and size of the tumor(s) and its effects on the local (abdominal) environment.

- Gastrinoma (p 696)

Gastrinoma may cause chronic vomiting due to gastritis or gastric ulcer disease that is unresponsive to routine therapy.

## NUTRITIONAL

- **Food intolerance\* (p 669)**

Food intolerance is a non-immune-mediated condition associated with intermittent diarrhea or vomiting, with no pattern or association with eating, and it resolves when the food source is changed to omit the offending substance from diet. The clinical significance of food intolerance is unknown relative to other causes of vomiting because of the difficulty in obtaining a definitive diagnosis.

## IMMUNOLOGIC

- **Food allergy (dietary hypersensitivity)\* (p 667)**

Chronic vomiting is a common clinical presentation in cats with food allergy, and cutaneous signs may also be present.

- **Idiopathic inflammatory bowel disease (IBD)\* (p 671)**

IBD causes chronic, intermittent or recurrent vomiting, anorexia, weight loss and diarrhea in cats. It is a diagnosis of exclusion and requires histopathologic confirmation.

## INFLAMMATORY

- **Chronic pancreatitis\*\* (p 673)**

Pancreatitis in cats is often **not** associated with vomiting, especially chronic pancreatitis. The most common signs are anorexia, lethargy and weight loss.

- Gastritis/gastric ulcer disease (p 679)

This is usually associated with an acute onset of vomiting, but low-grade gastritis may result in intermittent signs that do not prompt immediate attention.

## INFECTIOUS:

### Viral:

- Feline viral diseases (FeLV, FIV, FIP) (p 684)

Vomiting is not a common presenting sign for any of the primary feline viral diseases, but is associated with FeLV or FIV primarily secondary to the systemic disease that is occurring as a result of the infection. In cats with FIP, vomiting may be associated with focal granuloma formation in the GI tract or abdomen or may occur secondary to systemic disease (hepatic, CNS, renal). Other common clinical signs are anorexia, lethargy, fever or dyspnea.

### Bacterial:

- **Helicobacter spp.\* (p 682)**

The true importance of *Helicobacter* organisms in cats as a cause of chronic gastritis and vomiting is still not known, but there have been increasing numbers of cats with chronic gastritis and presence of these organisms that respond to appropriate therapy.

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### Protozoal:

- **Toxoplasmosis\* (p 686)**

Intermittent vomiting is a common sign in infected cats, but will occur in conjunction with other signs of systemic disease (respiratory, CNS, ocular).

### Fungal:

- **Histoplasmosis\* (p 685)**

GI histoplasmosis is less common than respiratory or disseminated disease, but vomiting will still often be observed, especially when there is hepatic involvement. The most common signs are respiratory (e.g. coughing or signs of respiratory distress) or lethargy, anorexia and weight loss associated with disseminated disease.

### Parasitic:

- **Physaloptera (p 690)**

Chronic intermittent vomiting may be associated with undiagnosed infection with the stomach worm *Physaloptera*.

- **Heartworm disease (*Dirofilaria immitis*) (p 691)**

Dirofilariosis is a disease with primarily regional importance. Cats with heartworm disease may not have signs of respiratory or cardiovascular disease (e.g. coughing or changes in breathing pattern), but instead present with intermittent vomiting, anorexia or weight loss.

### Toxin/drug:

- **Pharmacologic (drug-associated vomiting)\*\* (p 678)**

Drugs are a common cause of acute vomiting in cats. However, chronic vomiting may occur in cats that are on long-term drug therapy (e.g. chemotherapy, theophylline, etc.).

- **Lead poisoning (p 692)**

Lead poisoning is an uncommon cause of vomiting in cats, as they are generally more fastidious in their eating habits. In cats ingesting lead-containing materials, both gastrointestinal (vomiting, diarrhea, anorexia) and neurologic (seizures, ataxia, megaesophagus, tremors) signs are typical.

## INTRODUCTION

### MECHANISM?

**Vomiting** occurs when the **vomiting center** in the medulla is stimulated, which can occur by several mechanisms:

- Effect of blood-borne toxins or drugs.
- Afferent impulses from the cerebral cortex, chemoreceptor trigger zone (CRTZ), or receptors in the pharynx or abdominal viscera.

The **CRTZ** is stimulated by:

- Blood-borne toxins or drugs.
- Impulses from the vestibular apparatus.

**Vomiting is an active process** that must be distinguished from regurgitation.

Vomiting in cats is associated with **abdominal muscle contraction, considerable muscular effort and anxiety** prior to the event.

Vomit may consist of undigested food material (if swallowed whole), partly digested or even liquid, and may be clear, yellow (bile stained) or brown (food colored).

**Vomit is not typically tubular in form**, nor does it contain large amounts of white frothy material.

**The pH of vomitus is usually acidic** (pH < 4), **but may be neutral** (pH 7) if duodenal content is present (bile reflux).

## WHERE?

Vomiting may be associated with **gastrointestinal disease or extra-intestinal tract diseases**.

Gastrointestinal **disease involving the stomach and/or small intestine** causes vomiting.

Vomiting is **rarely associated with primary colonic disease**.

Extra-intestinal causes of vomiting include:

- **Hepatobiliary disease.**
- **Pancreatic disease** (e.g. pancreatitis, neoplasia).
- Renal disease causing acute or chronic **renal failure**.
- CNS disease (e.g. vestibular disease, encephalitis, seizure disorders, neoplasia).
- Metabolic diseases (e.g. **hyperthyroidism**, ketoacidotic diabetes mellitus or Addison's disease).
- Cardiomyopathy or congestive heart failure.
- Systemic illness or infection affecting the CRTZ or cortex (e.g. septicemia).

## WHAT?

**Primary gastrointestinal diseases** that cause chronic vomiting include:

- Parasites (e.g. physaloptera, etc.).
- Infectious diseases (e.g. *Helicobacter*).
- Inflammatory diseases (e.g. IBD, gastritis or gastric ulcer disease).
- Neoplasia.
- Mechanical (e.g. antral pyloric hypertrophy/stenosis, obstruction, intussusception).
- Dietary disturbances (e.g. food intolerance or food allergy).

**The most common causes of chronic vomiting are: dietary** (indiscretion or food intolerance), **neoplastic diseases, metabolic or extra-intestinal disturbances** (e.g. renal, hepatic or pancreatic disease) and **inflammatory diseases** (e.g. IBD).

**Diagnosis** is based on the minimum data base, multiple fecal examinations, survey or contrast radiography, radionucleotide or scintigraphic studies (e.g. thyroid disease, motility studies, evaluation for portosystemic shunts, vascular diseases, etc.), ultrasound examination, endocrine testing, evaluation of serum TLI, cobalamin/folate assays, titers for various infectious agents, neurologic examination and diagnostics (e.g. CSF,

computed tomography, etc.), endoscopic examination and biopsy or surgical exploratory with biopsy.

**Many diseases that cause chronic vomiting are diseases from which complete recovery is not possible** (e.g. IBD, food allergy, motility disturbances, chronic renal disease, etc.) and thus, **long-term therapy including dietary or pharmacologic treatment will be necessary**.

In some cases, the prognosis will be very guarded to poor for long-term survival (e.g. neoplasia, cardiomyopathy), and this should be discussed with the owner.

**Occasional vomiting is considered a normal phenomenon in cats**, both for removal of ingested hair, but also as a protective mechanism following consumption of new or unusual foods. This should be taken into consideration when evaluating a cat for chronic vomiting.

## DISEASES CAUSING SIGNS OF CHRONIC VOMITING (>3 WEEKS DURATION)

### FOOD ALLERGY (DIETARY HYPERSENSITIVITY)\*\*\*

#### Classical signs

- Chronic vomiting, usually < once per day, immediately to > 12 h after eating.
- Diarrhea is less frequent and more often large bowel in character.
- Weight loss.
- Dermatological signs in some cats (miliary dermatitis, pruritus).

### Pathogenesis

**Food allergy is an immunologic reaction usually to the protein (or glycoprotein) component of food.**

Food allergy may also be associated with dermatological signs.

Approximately 25% of cats with GI signs from food allergy also have dermatologic signs consisting of pruritus, miliary dermatitis and alopecia.

The pathogenesis of food allergy is poorly understood, but is thought to be a combination of two main

mechanisms. Firstly, direct toxicity caused by the ingestion of the food causing **release of histamine and other vasoactive amines**. Secondly, indirect effects are mediated via an amplification system, which responds to the food by releasing mast cell products, the production of **eicosanoids** and other inflammatory mediators, initiation of the kinin cascade, and other events that result in the clinical syndrome.

## Clinical signs

**Signs associated with food allergy are non-seasonal**, affect a wide range of ages, breeds and both sexes, and are present in one or all three of these systems: the gastrointestinal tract, the skin or the respiratory tract.

**Cats with food allergy may have vomiting/diarrhea, anorexia and weight loss**, but may also have **dermatologic signs**, which may include rodent ulcers, eosinophilic plaques, miliary dermatitis, otitis externa or generalized pruritus, alopecia and erythema. A combination of GI and dermatologic signs should raise the index of suspicion of a food-related problem.

**Head and neck pruritus** appears to be especially **common in cats with food allergy**.

In some cats, food allergy may be associated with systemic signs such as feline asthma, rhinitis, stomatitis or other respiratory tract signs. However, this is poorly documented.

In a study of cats with documented food sensitivity (food intolerance and food allergy), there was a history of vomiting (56%), diarrhea (25%), or vomiting and diarrhea (19%).

Vomiting usually occurred less than once daily, a few minutes to > 12 h after eating and most commonly consisted of bile.

Large bowel signs (mucus and fresh blood in feces or excessive straining to defecate) were slightly (57%) more common than small bowel signs. Flatulence occurred in 38% of cats.

Weight loss occurred in 70% of cats.

Appetite was variably affected and either normal, increased or decreased.

Some cats were reported as irritable (38%) or lethargic (25%).

Dermatologic signs occurred in 25% of cats and consisted of miliary dermatitis, pruritis and alopecia.

## Diagnosis

The **diagnosis of food allergy** is confirmed by performing **elimination/challenge trials**. However, **the diagnosis of food allergy may require a more specific approach to the elimination/challenge trials than is possible with commercially available diets** because of the difficulty in removing all sources of allergy from commercial diets.

The best **elimination diet** for making a definitive diagnosis **in these cases is a home-prepared diet containing novel protein and carbohydrate source**, e.g. lamb, venison, duck, kangaroo, crocodile or ostrich with potato or rice (and added vitamin and mineral supplementation).

Gastrointestinal signs usually resolve within 1–3 weeks but dermatologic signs may require that these diets be **fed a minimum of 6–8 weeks or 2 weeks after all symptoms resolve**.

The cat should then be changed to a **hypoallergenic commercial cat food** (e.g. IVD Limited diets, Hill's d/d diet or z/d diet, Royal Canin's Selected protein diets, or Eukanuba's Response Formula feline) containing food ingredients the cat has been successfully tested with or never exposed to.

**Skin tests are inadequate for diagnosis of food allergies** because they have a high incidence of false positives and negatives, and are only useful in determining which adverse reactions to foods have an IgE-mediated pathogenesis.

**Serum radioallergosorbent (RAST), ELISA**, or tests that are variations on the theme, are all available commercially to **test for the presence of antigen-specific IgE in the serum**. However, the difficulties present with skin testing still exist, i.e. non-IgE-mediated and delayed hypersensitivity reactions will also be missed. Not all food allergies cause systemic effects or cause increased systemic levels of IgE and so will be negative using serum IgE testing. Findings of a recent study suggest that type I hypersensitivities account for only 25% of gastrointestinal food sensitivities in cats. Gastroscopic food sensitivity testing has been tested in humans and

dogs, but not in cats, and its utility remains to be established for the routine diagnosis of food hypersensitivity.

## Differential diagnosis

The main differential diagnosis for food allergy is food intolerance and they are usually clinically indistinguishable.

Cats with food allergy causing **dermatologic signs** must be differentiated from cats with atopy, flea allergy dermatitis, psychogenic alopecia or insect bite hypersensitivity.

The **gastrointestinal signs** of either syndrome can mimic many other causes of chronic GI disease, including gastritis, pancreatitis, and small bowel diseases such as IBD or colitis. The severity and duration of each event depends on the amount of antigen ingested, the immune response and the sensitivity of the patient. The more severe the presentation, the more differentials that must be included, e.g. neoplasia, extra-intestinal disease, etc.

## Treatment

Treatment is initiated during the diagnostic phase by **feeding the elimination diet**. The key to effective therapy is to **find the offending agent(s) and remove them from the diet**.

**Corticosteroids may provide partial relief**, especially where type I hypersensitivity reactions are involved, but in general are not effective in maintaining remission or symptomatic relief for cats with food allergies.

**Antihistamines have not been proved effective** in preventing the gastrointestinal symptoms associated with food allergy.

Some animals will eventually develop allergies to components in the elimination diet. In these cases, feeding diets containing protein hydrolysates will be most effective. Unlike regular protein molecules, the molecular size of these hydrolysates is too small to crosslink IgE bound to mast cells.

## Prognosis

The prognosis for control of food allergy is excellent if the offending agent(s) can be identified and eliminated from the diet. However, in some cases, that is not

possible and affected animals will have recurrent or persistent clinical signs.

## FOOD INTOLERANCE\*\*\*

### Classical signs

- Chronic vomiting, usually < once per day, immediately to >12 h after eating.
- Diarrhea is less frequent and more often large bowel in character.
- Weight loss.
- Dermatological signs in some cats (miliary dermatitis, pruritus).

## Pathogenesis

**Food intolerance is a non-immunologic reaction to a substance or multiple substances in food.** This reaction can occur to proteins or other food components, but it can also be associated with food coloring, additives, preservatives or flavorings. Naturally occurring chemicals such as amines, salicylates and monosodium glutamate are present in food, especially highly flavored rich food.

These distinctions are **difficult to distinguish clinically**.

Food sensitivity may also be associated with dermatological signs.

Approximately 25% of cats with GI signs from food sensitivity also have dermatologic signs consisting of pruritus, miliary dermatitis and alopecia.

The pathogenic mechanisms of food intolerance are poorly understood, but a combination of direct toxicity caused by the ingestion of the food causing **release of histamine and other vasoactive amines**, and indirect effects that are mediated via an amplification system which responds to the food by releasing mast cell products, production of **eicosanoids** and other inflammatory mediators, initiation of the kinin cascade, and other events that result in the clinical syndrome.

## Clinical signs

Cats with **food intolerance** traditionally have been thought to have **gastrointestinal tract signs only**, because the syndrome is not an immunologic reaction.

However, humans with food intolerance may have dermatologic signs, mouth ulcers and other systemic signs. In cats, **vomiting, diarrhea, weight loss, poor coat condition and lethargy** are the most frequently observed clinical signs. It is unknown whether dermatologic signs also occur with food intolerance in cats.

The most common clinical signs of food intolerance are the same as food allergy, i.e. vomiting and/or diarrhea. The vomiting is often intermittent and may not necessarily follow any particular pattern in relation to eating. If diarrhea occurs, it is probably more likely to be large bowel in character (mucus and fresh blood in feces or excessive straining to defecate) than small bowel (low frequency, loose to watery feces passed without straining or mucus).

Weight loss may occur, along with a change in appetite.

## Diagnosis

The diagnosis of food intolerance is confirmed by performing elimination/challenge dietary trials.

An elimination trial (either using commercial hypoallergenic diets or premium diets that have reduced food colorings, preservatives, additives, etc.) of 4–6 days should be embarked upon first before proceeding to a more specific food elimination trial requiring controlled food preparation (single novel protein and carbohydrate source). Gastrointestinal signs usually resolve quickly. Vomiting may cease immediately and diarrhea usually within 3 days. Recrudescence of GI signs with challenge usually occurs within 4 days but may take up to 10 days with dermatologic signs.

## Differential diagnosis

**Food intolerance and food allergy are clinically indistinguishable** as they both present with the same clinical signs and are usually highly diet responsive.

**Other chronic causes of GI signs** include gastritis, pancreatitis and small bowel diseases such as IBD or colitis. As a general rule, more severe clinical presentations require a more thorough clinical workup to eliminate a wider range of differential diagnoses, e.g. neoplasia and extra-intestinal disease.

## Treatment

Food intolerance is highly diet-responsive once the offending agent(s) has been identified. This is usually done by using an elimination diet.

Since the condition is not immunologically mediated, drugs that modify or suppress the immune response are unlikely to be beneficial.

## Prognosis

Once the inciting food(s) has been eliminated from the diet, food intolerance has an excellent prognosis.

## HYPERTHYROIDISM\*\*

### Classical signs

- Weight loss.
- Polyphagia.
- Vomiting and/or diarrhea.
- Hyperactivity.
- Poor grooming habits.

See main reference on page 304 for details.

## Clinical signs

**Weight loss despite a voracious appetite (polyphagia)** is the most common owner complaint.

**Vomiting and polyuria/polydipsia** occur in a third of cats.

Other signs that occur less commonly are restlessness or irritability, poor grooming habits, decreased appetite, lethargy, panting or dyspnea and large bowel diarrhea.

**The most prevalent physical exam findings** are a palpable thyroid nodule, thin body condition, tachycardia or heart murmur and hyperactivity or irritability.

There is **no breed or sex predilection** for hyperthyroidism, but **most (90%) are old cats (> 10 years)**. Cats < 6 years of age are rarely affected.

## Diagnosis

**Presumptive diagnosis** is often suggested by the **age, history and clinical presentation**.

**Hematology changes** include **erythrocytosis, and a stress leukogram (leukocytosis, neutrophilia, lymphopenia).**

Serum biochemistry profile changes: **increases in liver enzyme activities (ALT, SAP, GGT, LDH, AST) occur in greater than 50% of cases**, hyperglycemia and pre-renal azotemia are less common. Hyperphosphatemia and hyperbilirubinemia are uncommon (< 10% of cats).

The **urine specific gravity will usually be > 1.035 (>50%)**, but cats with polydipsia/polyuria or underlying renal disease will have unconcentrated urine (urine SG < 1.035).

**Definitive diagnosis** is made by finding **elevated serum thyroxine (total T<sub>4</sub>) levels** in most cats (98%).

**In a few cats the total T<sub>4</sub> is in the upper half of the normal range** and measurements of **free T<sub>4</sub>, a T<sub>3</sub> suppression test** (useful for diagnosis of mild hyperthyroidism, but takes 3 days), **TRH stimulation test** (performed over 4 hours, side effects common from TRH administration) or **radionuclide (<sup>99</sup>Tc, pertechnate) imaging** examination (requires special equipment and isolation for 24 hours) **may be required to make the diagnosis.**

### Differential diagnosis

Diabetes mellitus, chronic renal failure, liver disease are common extra-intestinal causes of vomiting, weight loss and polyuria/polydipsia that must be differentiated from hyperthyroidism.

Primary GI diseases that may cause similar signs are food intolerance/food allergy, IBD and gastrointestinal neoplasia.

### Treatment

Medical therapy with anti-thyroid drugs that inhibit the synthesis of thyroid hormones, such as methimazole or carbimazole (2.5–5 mg/kg q 8–12 h, PO) are commonly used treatments. However, these drugs must be administered daily and have side effects which are potentially problematic. Vomiting and anorexia are relatively common side effects, while hematologic abnormalities are rare.

Calcium ipodate (15 mg/kg PO q 12 h) decreases serum T<sub>3</sub> (but not T<sub>4</sub>) concentrations acutely, but the effect is

only short term. Thus, it is not useful in long-term management of the disease.

Adjunctive therapy for hyperthyroid heart disease: beta-adrenergic receptor-blocking agents (propranolol, atenolol for heart rate control, and furosemide, calcium channel antagonists and ACE-inhibitors as needed for heart failure (see page 132).

Surgical thyroidectomy is a highly effective treatment, especially for unilateral disease, but requires an anesthetic procedure and careful technique to preserve the parathyroid glands. If bilateral thyroidectomy is required, the patient will require oral thyroid hormone supplementation. Recurrence of hyperthyroidism in the remaining gland or tissue is common.

Radioiodine therapy is the simplest, safest and most effective therapy for the majority of cats, except cats with chronic progressive renal disease/failure and cats that will not eat in confinement. The major disadvantage is the requirement for special facilities for use of radioisotopes and the cat cannot be handled for 7–21 days. This makes fluid therapy difficult if the cat goes into renal decompensation during that time, or requires other medication.

## IDIOPATHIC INFLAMMATORY BOWEL DISEASE (IBD)\*

### Classical signs

- Vomiting with or without diarrhea.
- Weight loss and anorexia or decreased appetite.

### Pathogenesis

Inflammatory bowel disease (IBD) is an **idiopathic inflammatory disease** of the feline GI tract characterized by **infiltration of the lamina propria and mucosa with inflammatory cells** of various types (lymphocytic, plasmacytic, granulocytic, granulomatous, eosinophilic or combinations of these).

**A lymphocytic/plasmacytic infiltration is the most common lesion in cats.**

The **etiology is unknown** but believed to involve genetic, immunologic, dietary, bacterial and mucosal factors.

**Lymphocytes and plasma cells are normal components of the feline GI tract**, and increased numbers in the GIT occur in response to many infectious agents. **Differentiation of feline IBD from other diseases requires a complete examination to rule-out other causes for the infiltration.** In IBD, there should also be evidence of **mucosal disease** (e.g. erosion, villous blunting, loss of normal structure in other ways, loss of normal function).

**Eosinophilic IBD is rare in cats**, and in some cases it is found in association with **hypereosinophilic syndrome** (infiltration of eosinophils in many body tissues including liver, spleen, lymph nodes, etc.).

Other forms of IBD, such as granulomatous and neutrophilic IBD are also rare in cats.

**Most forms of IBD involve the small intestine**, sparing the stomach and colon, however, enterocolitis and gastritis may also occur.

Recently **an association has been observed between cats with idiopathic IBD and concurrent cholangitis and pancreatitis.** The true relationship between these observations and clinical IBD is not known.

## Clinical signs

**Vomiting and weight loss are the most common signs**, but diarrhea and anorexia are also frequently observed signs.

**Middle-aged (> 4 years) cats are most frequently affected**, but all ages have been reported to have IBD.

There is **no breed or sex predilection**, but purebred cats may be at increased risk compared to DSH cats.

If there is concurrent cholangitis or pancreatitis, cats may also present with icterus, abdominal pain or fever.

## Diagnosis

**The diagnosis is based upon several factors:** (1) **histologic infiltration** of inflammatory cells, usually lymphocytes and plasma cells in the GI tract; (2) the presence of inflammatory cells is associated with **mucosal abnormalities and functional disturbances**; (3) there are **no other identifiable causes for the infiltration** identified; and (4) the signs of the disease are **chronic** (> 3 weeks in duration).

**It is especially important to rule out parasitic, infectious, dietary and extra-intestinal (e.g. hepatic, renal or pancreatic) causes of disease**, as these may cause similar lesions to IBD.

**The only means of ruling out dietary causes of vomiting is via an elimination trial**, which must be conducted using an elimination diet prior to making a diagnosis of IBD.

**Endoscopic examination and biopsy is the most common procedure** used to obtain the diagnosis. **Multiple (6–8), good-quality** (containing submucosa and properly oriented) **biopsies should be obtained from the small intestine and stomach.** Biopsies should be taken from all regions of the stomach and lymphoid follicles should be avoided when obtaining biopsies from the duodenum. **Full-thickness biopsies obtained surgically are also acceptable** if endoscopy is unavailable, and equal care should be taken to assure biopsy quality (proper handling to minimize artifact and orientation on the biopsy sponge to prevent curling and crush artifacts).

**The major difficulty associated with endoscopic diagnosis of IBD is differentiation of IBD from lymphoma**, which will not be possible if the biopsy samples are not deep enough (contain submucosa) or if they have significant mucosal artifacts that prevent adequate assessment of mucosal abnormalities. In some cases where histologic differentiation is impossible, immunocytochemistry techniques may have to be employed to distinguish the cell origin (e.g. lymphoma cells tend to be monoclonal, while lymphocytes from cats with IBD will be polyclonal).

**Non-specific laboratory abnormalities that may be found in cats with IBD include: hypoproteinemia, hyperglycemia** (stress induced), **hypokalemia, elevated liver enzyme activities and a stress leukogram.**

**Serum cobalamin and folate levels may be decreased in cats with IBD** due to malabsorption.

**Radiography and ultrasonography generally do not assist with the diagnosis**, but are important in ruling out other causes of the clinical signs, e.g. neoplasia.

## Differential diagnosis

**Neoplasia (lymphoma is of special mention because it may have a similar histologic appearance to IBD).**

**Food intolerance or hypersensitivity** (food allergy).

**Parasitic infections of the GI tract**, especially giardia, which are difficult to diagnose.

**Infectious diseases** such as *Campylobacter*, salmonellosis, *Clostridium*, etc.

**Extra-intestinal causes of vomiting** such as hyperthyroidism, uncontrolled diabetes, renal failure and pancreatitis.

## Treatment

In most cats, a **combination of dietary modification and pharmacologic therapy** (anti-inflammatory, immunosuppressive) is successful in controlling the clinical signs.

**Dietary therapy** involves three main strategies: (1) low-residue, highly digestible diets; (2) hypoallergenic, elimination diets aimed at controlling food allergies; or (3) high-fiber diets for cats with IBD that primarily involves the large bowel.

The mainstay of **pharmacologic therapy** in cats with IBD is **prednisone (2–4 mg/kg PO q 12–24 h)**. This dose is tapered after the first 2 weeks to the lowest dose that will maintain remission. Some cats can be weaned off prednisone after 3–6 months and maintained on a controlled diet (or diet with metronidazole), while others will require life-long steroid therapy.

In **refractory cases**, dexamethasone (0.25 mg/kg PO q 12–24 h) may be used as well, but is not suitable for alternate day therapy because of the long half life.

**Metronidazole** is often added (10–15 mg/kg PO q 12 h) to the treatment plan because of the anaerobic, antiprotozoal and immunomodulating effects of the drug.

**Other antibiotics that may be useful include tetracycline** (20 mg/kg PO q 8 h), doxycycline (5–10 mg/kg PO q 12 h) or **tylosin powder** (10–20 mg/kg PO q 12 h).

In cats that do not completely respond to corticosteroids or that become refractory to standard treatment approach, **chlorambucil** (1 mg PO q 3 days) or **azathioprine** (0.3–0.5 mg/kg PO q 2–3 days) can be added to the regimen. Toxicity (leukopenia) is a major risk with azathioprine use, and should be monitored by hemograms every 2–3 weeks.

## Prognosis

**Guarded.** Cats will frequently respond well to dietary and pharmacologic therapy, but relapses are common and long-term management is required. There is no known cure for IBD and owners should be educated about the nature of the disease to encourage compliance and understanding.

## CHRONIC PANCREATITIS\*

### Classical signs

- Lethargy.
- Anorexia.
- Vomiting is observed in less than 30% of cases.

See main reference on page 639 for details (The Cat With Signs of Acute Vomiting).

## Clinical signs

**Lethargy and anorexia** have been the most commonly observed clinical signs.

In general, **vomiting and anterior abdominal pain occur less frequently.**

Signs may be protracted, with low-grade vomiting occurring in some cats, while others have acute signs.

## Diagnosis

A certain **degree of clinical suspicion** is required to make the diagnosis of pancreatitis in cats because the signs are so vague.

Cats with chronic pancreatitis may develop **secondary complications** such as exocrine pancreatic insufficiency, diabetes mellitus, obstruction of the common bile duct or cholangitis.

**Hematology and serum chemistry profiles are non-diagnostic**, but may show dehydration, electrolyte imbalances, elevated liver enzymes, mild hyperbilirubinemia or a leukocytosis.

**Serum lipase and amylase values are not helpful**, as they may be elevated, normal or low in normal cats or cats with pancreatic disease.

**Imaging studies are the most useful** way to obtain a presumptive diagnosis of pancreatitis. **Radiographs of the anterior abdomen** may reveal evidence of local peritonitis, gas in the duodenum or anterior jejunum, or a mass. **Ultrasound examination remains the most reliable technique to evaluate the pancreas** and determine the presence of swelling, abscess or fibrosis. It is important to note that **ultrasonographic changes in chronic pancreatitis persist for months**.

Elevations in serum **trypsin-like immunoreactivity (TLI) assay** have been suggested as a means of detecting pancreatitis, but this **has not been shown to be consistently accurate** either, and is of less use in chronic pancreatitis.

The assay for feline pancreatic lipase (fPLI) has been developed and is a sensitive and highly specific test for acute pancreatitis. However, in cats with chronic pancreatitis, the fPLI may be normal, or mildly increased but below levels diagnostic for pancreatitis, which can make test interpretation difficult.

## Differential diagnosis

The list of problems causing anorexia and lethargy in cats is lengthy and includes multiple organ systems and abnormalities.

## Treatment

**Nothing should be fed for 1–2 days if the cat is vomiting** until the vomiting can be controlled. However, a fast of longer than 3 days is to be avoided if at all possible in most cats.

**Intravenous fluid therapy** should be administered, using a balanced electrolyte solution (either replacement or maintenance fluids) to maintain hydration, correct pancreatic ischemia and reduce the risk of complications of pancreatitis.

**Anti-emetics** (e.g. metoclopramide 0.1–0.2 mg/kg) may be used if the cat is vomiting.

Cats with severe pancreatitis may be unable to eat for several days (3–5), thus, **parenteral or partial enteral nutrition** will be necessary to prevent development of malnutrition, feline hepatic lipidosis and impaired immunity.

Steroid therapy may be indicated in cats with concurrent IBD or cholangitis.

## HEPATIC DISEASES (HEPATIC LIPIDOSIS, CHOLANGITIS, HEPATIC CIRRHOSIS, PORTOSYSTEMIC ANOMALIES)

### Classical signs

- Lethargy or depression, vomiting, anorexia and weight loss are common signs.
- Icterus, ascites, hepatomegaly or microhepatica are variable.

See main references on page 421 for details (The Yellow Cat or Cat With Elevated Liver Enzymes).

### Clinical signs

**Vomiting occurs primarily because of the gastritis that occurs secondary** to changes in blood flow and release of GI hormones, and because of mediator release (stimulation of the vomiting center) secondary to presence of toxins from decreased hepatic clearance or function.

Liver diseases often present with the relatively non-specific signs of **anorexia, weight loss, vomiting, and lethargy or depression with or without icterus**.

Other signs include **ascites, edema formation, bleeding disorders due to deficiency of coagulation factors or DIC**, and in cases with severe hepatic failure, signs of **hepatoencephalopathy** (dementia, personality changes, seizures, stupor or coma). Prolongation of clotting times is quite common in cats with hepatopathy.

### Diagnosis

**Icterus** (hyperbilirubinemia) may be caused by **pre-hepatic (hemolysis), hepatic or post-hepatic (bile duct obstruction)** diseases which all must be considered.

The **hemogram** is important in **differentiating hemolysis from hepatic causes of icterus**. **Anemia is a common problem** and may be either regenerative (secondary to blood loss or hemolysis) or non-regenerative (e.g. anemia of chronic disease, bone marrow suppression). Typically the anemia of chronic disease is

less severe (PCV > 20), than that seen with hemolysis or severe blood loss.

Target cells (leptocytes) on the blood smear are also suggestive of liver disease.

Serum biochemical profile results will be supportive of the diagnosis, with the most common findings being **elevated liver enzymes, hyperbilirubinemia**, decreased BUN, decreased albumin and total protein, hypocholesterolemia, and electrolyte disturbances consistent with vomiting (hypokalemia, hypochloridemia, hyponatremia).

A **serum bile acid assay** is the most reliable, easiest to perform and most readily available liver function test. A **pre- and 2-hour post-prandial assay should be submitted** to evaluate liver function. Other function tests include blood ammonia and bromosulphophthalein (BSP) retention.

Measurement of sulfated bile acids in urine is a newly developed test to diagnose liver disease. In many cats that will not eat, obtaining a serum post-prandial bile acid sample is difficult, and this test may provide additional information about the status of liver function in inappetent cats.

Imaging studies (radiographs, **ultrasound**) are used to assess liver size, morphology and structure, and may be used to obtain **aspirates or biopsies** of liver via ultrasound-guided techniques.

In all cases of suspected liver failure, **evaluation of coagulation function is essential**, not only for therapeutic purposes (e.g. determining the presence of DIC, coagulopathies), but also prior to obtaining tissue for biopsy.

The **definitive diagnosis** typically requires **histopathologic examination** of liver tissue, however, in cases of suspected vascular anomalies, a venous portogram or portal scintigraphy is necessary to define the abnormal vascular structures.

## Differential diagnosis

**Icteric cats:** Immune-mediated hemolytic anemia, pancreatic or gastric abscess or tumor obstructing common bile duct, hemolytic anemia secondary to red cell parasites, primary liver diseases (hepatic lipidosis, cholan-

gitis, and hepatic lymphoma or carcinoma) are the most common diseases.

**Cats with ascites:** Ascites may be caused by pre-hepatic (cardiac), hepatic or post-hepatic (GI lymphatic obstruction, protein-losing enteropathy, severe protein-losing nephropathy or mesenteric hypertension (torsion) or venous thrombosis or vasculitis), and these must be separated. **Ascites due to cardiac failure is very rare in the cat.** Hepatic disease, vasculitis from feline infectious peritonitis or lymphatic obstruction associated with neoplasia are the most common causes of ascites in cats.

## Treatment

Treatment is based upon **identifying the primary problem**. The reader is referred to the main reference (page 421) for details.

## GI LYMPHOSARCOMA\*\*

### Classical signs

- Weight loss.
- Intermittent or persistent vomiting.
- Small or large bowel diarrhea.
- Lethargy or depression.

## Pathogenesis

In the United States and other countries where **feline leukemia virus (FeLV) is prevalent**, **FeLV infection is the most common cause of all types of lymphoma except the alimentary form**, which is only associated with viremia 25–30% of the time. In areas **where FeLV infection is rare** (e.g. east coast of Australia), **most cats with lymphoma are FeLV negative**.

Some evidence suggests that the alimentary form of lymphoma arises from transformed multipotent lymphoid or monocyte precursors or from FeLV-transformed B-lymphocytes, and thus may still be associated with FeLV.

Alimentary lymphoma is the **second most common GI tumor** (adenocarcinoma is first), and occurs in **older cats** (> 8 years).

The **most common site** for lymphoma is the **jejunum or ileum**, with the duodenum being a rare site of occurrence. Lymphoma is the most common tumor found in the feline stomach (although it is still a rare disease), but is found unfrequently in the colon compared to adenocarcinoma.

Lymphoma can occur as a **solitary mass causing an obstructive lesion, or as diffuse disease** affecting many sections of the GI tract and occurring throughout all layers of the bowel wall.

## Clinical signs

The **most common clinical signs** associated with intestinal lymphoma in cats are **anorexia, lethargy and weight loss**.

**Vomiting and diarrhea are variably observed**, depending on the location of the lesion, the nature of disease (diffuse vs. a solitary mass) and duration of disease e.g. fever, hematemesis, icterus, ascites and melena.

Cats that are FeLV positive may also have clinical signs consistent with the disease in other locations (e.g. anemia, leukemia, etc.).

## Diagnosis

**Hematology may be normal or there may be an anemia of chronic disease** (that may be masked by dehydration) in cats that are FeLV negative. **FeLV-positive cats may have a wide variety of hematologic changes**, which should be further investigated with a bone marrow examination.

**Serum biochemistry profile values also may be within normal limits**, or there may be **increases in liver enzymes, hypercalcemia or hypoproteinemia**. Pre-renal azotemia and electrolyte disturbances may occur in cats with severe vomiting.

The **majority of cats (70–75%) are FeLV antigen negative**.

**Radiographs** may be normal or may reveal thickened bowel loops or a solitary mass lesion.

**Ultrasonography** is useful for localizing solitary masses, evaluating mesenteric lymph nodes (which are also typically involved), and for identifying thickened intestinal loops.

The **definitive diagnosis** is made by **histopathologic evaluation** of affected segments of bowel. **Ultrasound-guided aspirates** often will provide sufficient tissue sample for the diagnosis (intestinal wall, mass or lymph nodes).

If the lesion is in a location accessible by **endoscopy**, **biopsies** can be obtained by this method. However, **endoscopic biopsy samples can be easily misread as IBD or visa versa**, and the diagnosis can be missed if the lesion is present only in the submucosa.

If these diagnostic modalities are not available or provide inconclusive results, full-thickness biopsies obtained by surgical exploratory are usually definitive.

**Staging the disease** requires histologic evaluation of regional (mesenteric) lymph nodes, liver and spleen, radiographic evaluation of lungs and examination of bone marrow aspirates (if indicated) for evidence of metastasis.

## Differential diagnosis

The major differential is **lymphocytic plasmacytic IBD**.

Intestinal adenocarcinoma, foreign body, FIP, fungal or algal infections of the GIT, other intestinal tumors (e.g. mast cell tumor) and hyperthyroidism must all be considered.

## Treatment

**Chemotherapy is provided in stages:** induction of remission, intensification, maintenance and rescue. In general, **cats are more difficult to rescue** (compared to dogs) **once the cancer is out of remission**, which is why their survival times are shorter than dogs.

**A typical protocol used to induce remission is the COAP protocol:** cyclophosphamide (200–300 mg/m<sup>2</sup> PO q 3 weeks), vincristine (0.5 mg/m<sup>2</sup> IV q 1 week), cytosine arabinoside (100 mg/m<sup>2</sup> IV or SC for 2 days only), and prednisolone (50 mg/m<sup>2</sup> PO q 24 h for 7 days, then EOD). If remission is only partially achieved, intensification with doxorubicin (25 mg/m<sup>2</sup> IV q 3 weeks) or mitoxantrone (4–6 mg/m<sup>2</sup> IV q 3 weeks) can be tried.

**Maintenance chemotherapy** protocols may include continuation of the COAP protocol or use of the LMP protocol (chlorambucil, methotrexate, prednisone).

In cats with small cell (highly differentiated) lymphoma of the GIT, the combination of chlorambucil (20 mg/m<sup>2</sup> PO every other week) and prednisolone (20 mg/m<sup>2</sup> PO EOD), with or without methotrexate (2.5 mg/m<sup>2</sup> PO EOD) is often successfully used.

Other protocols that have been successfully used to treat feline lymphoma include COP (cyclophosphamide, vincristine and prednisone), VCM (vincristine, cyclophosphamide and methotrexate), or VCM plus L-asparaginase.

**Nutritional support should be used as needed** (especially in cats that are vomiting or not eating enough to maintain normal function).

**Control of secondary infections** is also sometimes necessary and should be implemented as required. In cats with alimentary lymphosarcoma, metronidazole is often used as the antibiotic choice.

## Prognosis

**FeLV status does not influence whether or not remission occurs, but is a significant predictor of survival**, with FeLV-negative cats having much longer survival times. FeLV-positive cats in stages III–V had no differences in survival than FeLV-negative cats, but in stage I–II, survival is 18 months versus 4 months, respectively, for negative cats and cats with the virus.

The **overall prognosis is guarded to poor** in cats with alimentary lymphoma. Most cats with alimentary lymphoma respond poorly to chemotherapy, but those that do respond may have a survival time of more than one year. Cats with a solitary mass in the GIT that can be resected have a better prognosis.

**Most cats with lymphoma treated with multiple agent chemotherapy** protocols are expected to live **3–9 months**, with 20% living longer than 1 year. **Untreated cats can only be expected to survive 4–8 weeks**. Cats that are **FeLV positive** also have **shorter survival times**.

**Survival does not appear to be influenced by the extent of the disease, anatomic location or clinical stage**, which is an important predictor in some types of lymphoma.

## INTESTINAL ADENOCARCINOMA\*\*

### Classical signs

- **Weight loss and lethargy or depression are the most prevalent signs.**
- **Intermittent to persistent vomiting or hematochezia develop as the tumor obstructs or ulcerates the epithelial surface.**

## Pathogenesis

The **most common non-hematopoietic tumor** of the feline GI tract is the adenocarcinoma.

Most intestinal adenocarcinomas occur in the **jejunum and ileum**, with the duodenum rarely affected. Colonic adenocarcinoma is uncommon and usually occurs in **very old cats (e.g. 16 years)**.

**Siamese cats** represented 70% of 225 reported cases of **intestinal adenocarcinoma** and had a mean age of 10–11 years.

**Domestic shorthair cats** had the most reported cases of **colonic adenocarcinoma**.

**Feline leukemia virus appears to have no role in the pathogenesis of this disease**, since all 28 cats in one study that were tested were found to be FeLV negative.

Adenocarcinomas occur either as **annular or intraluminal tumors**.

**Metastasis** of adenocarcinomas is typically **within the abdominal cavity and not to the lungs**.

## Clinical signs

The most commonly observed clinical signs are **anorexia, lethargy, weight loss and vomiting**.

**Hematochezia, melena or diarrhea** are primarily observed in cats with **colonic disease**.

In cats that are not eating and are vomiting persistently, dehydration may also occur. Fever and abdominal effusion are also late-stage findings associated with metastasis of the tumor.

## Diagnosis

**Hematologic and serum biochemical profiles are often normal.**

**Abnormalities may include:** hemoconcentration, non-regenerative anemia of chronic disease, electrolyte imbalances or pre-renal azotemia, hypoproteinemia or hypoalbuminemia due to GI protein loss, or elevated liver enzyme activities (metastatic disease).

**Radiographs of the thorax rarely reveal metastatic lesions.**

**Abdominal radiographs** may show a mass, enlarged lymph nodes or signs of an intestinal obstruction (e.g. dilated, air-filled bowel segment, abnormal gas patterns, or differences in bowel size or location relative to neighboring bowel). Some cats will have tumors with osseous **metaplasia (mineralization)** or **ascites** that will imply neoplasia. Contrast studies may be very useful in delineating intraluminal or annular lesions.

**Ultrasonography** is also very helpful in identifying mass lesions and their extent for staging, and can be used to direct **fine-needle aspirates or biopsies** for diagnosis.

In cats with colonic disease, **colonoscopic evaluation and biopsy may also be useful.**

**Surgical exploratory** is the best way to obtain the definitive diagnosis, get **accurate staging information**, and will also allow **resection of the affected segment** of bowel if that is indicated.

## Differential diagnosis

Intestinal foreign body or obstruction, other intestinal neoplasia, inflammatory bowel disease, vomiting and weight loss due to extra-intestinal disease (chronic renal failure, etc.), fungal or algal diseases of the intestine, and FIP granuloma should all be included in the differential list.

## Treatment

The **only reported and effective treatment** for intestinal adenocarcinoma in cats is **surgical resection.**

The **role of adjuvant chemotherapy** for feline intestinal adenocarcinoma has not been explored.

**Nutritional support may be necessary** in cats that do not eat enough to meet at least resting energy requirements ( $RER = 30 \times BW \text{ (kg)} + 70$ ) and cats with cancer may require additional calories ( $RER \times 1.5$ ).

**Cats with large colonic resections may require very highly digestible diets** to reduce the fecal volume and load on the colon.

## Prognosis

The **presence of metastatic disease is not necessarily a poor prognostic sign** in cats with adenocarcinoma, and should not be a disincentive for performing a surgical resection and anastomosis.

The **average survival for cats with the disease is 4–15 months** (with or without metastatic disease), but some cats live several more years following aggressive surgical resection.

## PHARMACOLOGIC (DRUG-ASSOCIATED VOMITING) \*\*

### Classical signs

- Vomiting observed after administration or ingestion of a drug.

See main reference on page 642 for details.

## Clinical signs

In some cases, vomiting is expected following drug administration (e.g. chemotherapy, xylazine), while in others it is not usual (e.g. tetracycline, erythromycin), and in others it represents toxicity or accidental exposure (NSAIDs, narcotics, digitalis).

The **signs observed will depend on the drug that is administered**, e.g. chemotherapy drugs may cause vomiting, anorexia, lethargy, hair loss, etc., while vomiting caused by chloramphenicol or erythromycin may be the only clinical sign.

## Diagnosis

Diagnosis is largely dependent upon obtaining an **extensive, accurate history** that includes a complete

history of drugs the cat is taking or has taken, toxins it has been exposed to and the potential for accidental or malicious poisoning.

Some **commonly used drugs known to cause vomiting in cats include** many, if not most, **antibiotics** (e.g. amoxicillin, cephalexin, enrofloxacin, tetracycline, erythromycin, metronidazole, clindamycin), **chemotherapy drugs** (dacarbazine, cisplatin, doxorubicin, methotrexate, cyclophosphamide), **methimazole**, **potassium bromide**, **glipizide**, and **antifungal drugs** (e.g. itraconazole, amphotericin).

In some cases, **determination of blood levels of a specific drug** will confirm the presence of a drug or toxin that was unknown or unexpected. Alternatively, hair and urine samples can also be used in some cases to determine the presence of metabolites of various drugs or chemicals.

**If the suspected exposure is recent, evacuation of the stomach and analysis of its contents** will also provide a means of determining the presence of drugs or chemicals.

### **Differential diagnosis**

Other acute causes of vomiting such as food intolerance/dietary indiscretion, parasitic diseases, infectious diseases, foreign bodies or gastritis due to other causes should be considered when the history is not helpful in identifying exposure.

### **Treatment**

Choice of treatment depends on whether the vomiting is expected (e.g. chemotherapy) and can be controlled with anti-emetics (e.g. metoclopramide or chlorpromazine), or is due to unexpected toxicity or accidental ingestion.

**Supportive care for toxicity or accidental ingestion** includes removal of stomach contents and gastric lavage, administration of activated charcoal to reduce absorption of contents, administration of gastric protectants as indicated (NSAID ingestion will cause gastritis), anti-emetics (metoclopramide or chlorpromazine), and fluid support to prevent or treat dehydration.

**In cats with vomiting due to intolerance** (e.g. tetracycline), changing the drug protocol to another that will be effective is generally all that is indicated.

## **GASTRITIS/GASTRIC ULCER DISEASE**

### **Classical signs**

- Frequent vomiting is typical, but intermittent vomiting will occur in some cats.
- Hematemesis or melena occurs with severe gastritis or bleeding ulcers.

See main reference on page 637 for details (The Cat With Signs of Acute Vomiting).

### **Clinical signs**

**Vomiting**, either frequently or intermittently, is the primary clinical sign. Severe gastritis and ulcer disease will result in **anorexia**, **abdominal discomfort**, **lethargy** or **weight loss**.

Cats with severe gastritis or ulcer disease may also have **hematemesis** or **melena** from mucosal bleeding.

### **Diagnosis**

**Endoscopic examination** of the gastric mucosa will often reveal the surface erosions or ulcers, however, **histologic examination** of the tissue is required to make a diagnosis of gastritis. Mild or chronic gastritis lesions may not have an abnormal mucosal appearance.

Gastric or duodenal ulcers can also be identified by **contrast radiography** and by **ultrasonography** in some cases, but these techniques are not as useful as endoscopy.

Most cats with gastritis will have a **normal hemogram and chemistry profile**. Cats with bleeding ulcers may have evidence of **acute** (non-regenerative if peracute, regenerative if recent) or **chronic blood loss** (may be either regenerative or non-regenerative depending on amount of hemorrhage).

### **Differential diagnosis**

*Helicobacter* spp. infection, parasites (*Physaloptera*, *Ollanus* spp.), IBD, neoplasia, gastritis due to other extra-intestinal diseases.

**In cats with ulcers that do not respond to standard therapy**, ulcer disease due to a **gastrinoma** should be

**ruled out.** Diagnosis of a gastrinoma is made histologically by finding a neuroendocrine tumor, usually in the abdomen, and by confirming the presence of **hypergastrinemia** (serum gastrin levels that are extremely elevated) not due to secondary causes.

## Treatment

**Specific treatment** is aimed at identifying and correcting the underlying cause.

**Non-specific treatment** for gastritis or gastric ulcers involves **reduction of gastric acid secretion, protection of the mucosa** to allow healing and **reduction of gastric retention or vomiting**. This is accomplished by:

**Histamine-2 blockers:** ranitidine (0.5–1.0 mg/kg PO q 12 h), famotidine (0.5–1.0 mg/kg PO q 12–24 h).

**Proton pump inhibition:** omeprazole (0.5–1.0 mg/kg PO q 24 h), note: this drug is difficult to dose in cats as it must be recompounded, but is the most effective acid suppression drug.

**Mucosal protection:** sucralfate (250–500 mg PO q 8–12 h) note: give at least 2 hours before giving H<sub>2</sub> blockers.

**Anti-emetics:** metoclopramide (0.1–0.2 mg/kg PO q 8–12 h) as needed to control vomiting, promote gastric emptying and reduce gastroesophageal reflux.

**Feed only highly digestible, low-fat foods** (e.g. Hill's i/d diet, Purina EN, Iams low-residue diet, etc.) **in small amounts**. In some cats, it is best to give them nothing per os until the gastritis/ulcer healing has begun and the vomiting is under control (1–3 days).

Hydration should be maintained with **IV or SQ fluids as needed**, if the cat is unable to eat or has severe vomiting.

## CHRONIC RENAL FAILURE\*

### Classical signs

- Polyuria/polydipsia.
- Weight loss.
- Anorexia.
- Lethargy.
- Poor/hair coat/unkempt appearance.
- Vomiting.

See main reference on page 235 for details (The Cat With Polyuria/Polydipsia).

## Clinical signs

**Polyuria/polydipsia, anorexia** or decreased appetite, and **weight loss** are the most common clinical signs of chronic renal failure.

An unkempt hair coat, decreased grooming and reduced overall activity level are also often observed.

Intermittent vomiting or diarrhea due to hypergastrinemia and uremia and uremic ulcers or stomatitis occur in severe cases.

**Polyuria** is profound until the late stages of the disease, when oliguria gradually develops with the progression to end-stage kidney disease.

## Diagnosis

History and physical examination findings are suggestive of the diagnosis in a geriatric cat.

**Hematology, serum biochemistry profile and urinalysis** will confirm the presence of **azotemia, isosthenuria** or poorly concentrated urine (1.015–1.035), **mild to moderate non-regenerative anemia**, dehydration may also be present, along with **hypokalemia, hyperphosphatemia**, and evidence of chronic metabolic acidosis (low total CO<sub>2</sub>).

## Differential diagnosis

Hyperthyroidism, diabetes mellitus, hepatic disease (hepatic lipidosis if cat is/was obese) and neoplasia (lymphoma, adenocarcinoma, etc.) are important differentials in an old cat with polyuria/polydipsia and weight loss.

## Treatment

**Supportive. Fluid therapy** as needed. **Sub-cutaneous fluids may be very important adjunctive therapy** in cats that do not drink enough or are vomiting, and have severe polyuria that results in dehydration and worsening azotemia.

**Dietary manipulation.** Feeding a diet that has a **moderate or low quantity of high-quality protein, reduced phosphorus, and relatively high in calories**, to maintain weight, decrease azotemia and reduce the effects of renal secondary hyperparathyroidism.

**Phosphorus binders** (e.g. amphoteg) may be needed when dietary manipulation no longer is effective in reducing serum phosphorus levels.

**Potassium supplementation** (potassium gluconate) is needed in most cats with chronic renal failure due to potassium wasting and total body potassium depletion that occurs.

**In cats with frequent vomiting, histamine-2 blockers** (famotidine 0.5–1.0 mg/kg PO q 24 h, ranitidine 0.5–1.0 mg/kg) may be helpful in reducing hyperacidity and gastritis that occurs secondary to azotemia and hypergastrinemia.

Cats in more advanced stages of renal disease may have a significant **non-regenerative anemia (PCV < 20)** due to the lack of erythropoietin production from the failing kidney, which may respond, for a limited period of time, to treatment with **recombinant human erythropoietin**.

## DIABETES MELLITUS\*

### Classical signs

- Polyuria/polydipsia.
- Weight loss.
- Polyphagia or anorexia.
- Lethargy, weakness and/or depression.
- Vomiting.

See main reference on page 236 for details (The Cat With Polyuria and Polydipsia).

### Clinical signs

Signs of **polydipsia, polyuria, polyphagia and weight loss** may be **present for months prior to diagnosis**.

Approximately **one-third of cats with diabetes mellitus have a history of vomiting**. An **acute onset of vomiting** may occur with **ketoacidosis**.

The **classical signs of ketoacidosis** include **polyuria, polydipsia, anorexia, weight loss, dehydration, vomiting, weakness and depression**.

In some cats, other clinical signs may include **peripheral nerve dysfunction (plantigrade stance), dementia or stupor/coma**.

## Diagnosis

The **clinical signs and history** of polyuria/polydipsia suggest that diabetes mellitus should be considered.

The **hemogram is non-specific**, hemoconcentration and a stress leukogram are common, however, some cats have an inflammatory leukogram secondary to infection (a common sequelae of unregulated diabetes mellitus).

A **serum biochemistry profile and urinalysis** will be **diagnostic**. The finding of **hyperglycemia with glucosuria with or without ketonuria, and in some cases bacteriuria/pyuria is typical**. Other abnormalities likely to be observed include elevated liver enzymes, hypokalemia (may be severe), hypophosphatemia (may be severe), hypomagnesemia, low total CO<sub>2</sub> values (supportive of acidosis), and azotemia (may be pre-renal or renal in origin).

**Other studies**, such as imaging, blood gas analysis, etc. are used to further characterize the disease relative to severity and cause (e.g. pancreatitis, etc.), but **should not be done at the expense of initiating appropriate and immediate therapy**.

### Differential diagnosis

The **differentials are numerous, based upon the history and clinical signs** (e.g. renal failure, hepatic disease, poisoning, neoplasia and inflammatory bowel disease) but a minimum data base will serve to confirm the presence of diabetes mellitus.

## Treatment

**Ketoacidotic diabetes mellitus can be a medical emergency**, as many cats will have severe metabolic acidosis, dehydration and electrolyte disturbances that are life threatening unless addressed.

**Fluid therapy** (0.9% NaCl or Normosol) is the essential, but must be used judiciously in conjunction with **replacement of potassium, phosphorus and magnesium**, which are often severely depleted. Serum potassium may rapidly decrease during initial therapy and should be monitored very closely (q 2–4 h).

**Insulin replacement** is instituted with a short-acting insulin (e.g. regular insulin, 0.1–0.2 U/kg, IV or IM q

2–6 h or as a constant rate infusion). **Frequent monitoring (q 1–2 h) of blood glucose concentrations is necessary** so that when the blood glucose level drops below 16.8 mmol/L SI (300 mg/dl), 5% dextrose is added to the fluids.

In cats that are not severely acidotic, and are still eating and not vomiting, insulin treatment may be instituted with NPH insulin (0.25–0.5 U/kg, SQ q 12 h), or where available PZI or glargine, with monitoring of blood glucose levels (q 2–3 h).

**Cats that have urinary tract or other infections** should be treated with appropriate **antibiotics**.

**Vomiting is controlled with metoclopramide** (0.1–0.2 mg/kg q 12 h) as needed.

Cats that are able to eat without vomiting should be fed a **highly palatable diet** (see page 238). **High-protein, reduced-carbohydrate diets** are advantageous in both thin and obese diabetic cats unless they have concurrent renal disease and need to eat a lower-protein diet.

## HELICOBACTER SPP.\*

### Classical signs

- The classical sign is vomiting.

### Pathogenesis

*Helicobacter* organisms known to **colonize the feline stomach** include: *H. pylori*, *H. felis*, and *H. bizzozeronii*. Other *Helicobacter* spp. that have been identified in the small intestine and liver of cats include *H. cinaedi* and *H. fennelliae*. The significance of these organisms and their ability to cause clinical disease is unknown.

*H. felis* is commonly found in the stomach of cats and may not be a feline pathogen, but *H. pylori*, which causes peptic disease in humans, is believed to also be associated with **gastritis in cats**, especially cats in **colonies or catteries**. *H. bizzozeronii* is the most common gastric colonizer in cats, and has experimentally produced chronic gastritis in kittens.

Infection is primarily transmitted via the **oral–oral route**. **Organisms are present in vomitus, oral secretions and saliva**, and may be transmitted via improperly disinfected dental equipment, endoscopes and

other instruments, or via contact with oral secretions directly.

Infection with *Helicobacter* organisms results in **increased infiltration of the mucosa with polymorphonuclear cells and mononuclear cells**, and is classified as **chronic gastritis**.

**Persistent infection is associated with increased development of gastric lymphoid follicles**, especially in the antrum, the location of heaviest concentration of organisms in most cats.

At this time, there does not appear to be a breed or sex predilection for infection or development of signs.

**The classical sign is chronic vomiting due to gastritis**, but **the incidence and true importance of *Helicobacter* spp. in gastritis in cats is not known**.

### Clinical signs

**Chronic vomiting, weight loss** and in some cases, pica are reported in clinically affected cats.

However, **these organisms will be present in clinically healthy animals**, so a direct cause-and-effect relationship cannot be established.

### Diagnosis

The diagnosis is **made by histologic examination of gastric mucosal tissue (usually obtained via endoscopy)**. Either a **special silver stain or modified Giemsa stain** must be used to identify the organisms.

**Culture and organism identification is also required to make a definitive diagnosis**, since not all spiral organisms are pathogens or *Helicobacter* spp. *Helicobacter* spp. require **special media and handling for successful culture in vitro**.

***Helicobacter* spp. produce urease** that can be used to provisionally diagnose their presence, by placing biopsy samples in urea broth or by using the **commercially available test (CloTest)**. However, **this test is neither sensitive nor specific for *Helicobacter* infection**.

**Serologic assays used in humans are not reliable for testing in cats** because of the cross-reactivity between other spiral organisms (commonly present in normal cats) and *H. pylori* (the suspected pathogen).

## Differential diagnosis

Chronic gastritis due to other causes such as hepatic disease, pancreatitis, chronic renal failure or IBD may result in a similar clinical presentation.

## Treatment

There have been various treatment regimens recommended for cats with *Helicobacter* infection, but none have been well substantiated.

**The triple therapy regimen** has been the mainstay of treatment for *Helicobacter* infection. This is bismuth (0.5–2 ml/kg PO q 4–6 h), metronidazole (62.5 mg PO q 24 h), and amoxicillin (15–20 mg/kg PO q 12 h). This regimen with the addition of famotidine (0.5–1.0 mg/kg PO q 24 h) has been recommended in refractory or severe cases.

Famotidine (0.5–1 mg/kg PO q 24 h), ranitidine (1–2 mg/kg PO q 12 h), or omeprazole (0.5–1.0 mg/kg PO q 24 h), in addition to antibiotic therapy have more recently been recommended.

Other antibiotics that may be used include tetracycline (22 mg/kg PO q 8 h), clarithromycin (7.5 mg/kg PO q 12 h), or azithromycin (5 mg/kg PO q 24–48 h).

Pepto-bismol can be used (1 ml/kg PO q 24 h) to control gastritis due to *Helicobacter* infection, but salicylate toxicity has occasionally been observed in individual cats.

The **duration of treatment for infected cats** has also not been well substantiated, but **14–28 days** has been the standard recommendation.

## Prevention

Unknown, but careful attention to cleanliness of endoscopes and dental equipment is advised.

## Public health

*H. felis* and *H. heilmannii* both are capable of colonization of humans and cats, but are not particularly pathogenic. *H. pylori* has not been isolated reliably from pet cats, so the zoonotic potential of *Helicobacter* in pet cats as a cause of human gastritis (a significant problem) has not been shown.

**Most epidemiologic studies do not incriminate pet contact with human infection**, and only contact with commercially reared cats has been shown to have any potential for risk, suggesting the possibility of a reverse zoonotic infection.

## HYPERCALCEMIA\* (MALIGNANCY, CHOLECALCIFEROL TOXICITY, CHRONIC RENAL FAILURE, HYPERPARATHYROIDISM, IDIOPATHIC HYPERCALCEMIA)

### Classical signs

- Signs vary with the cause of hypercalcemia.
- Anorexia, vomiting and weight loss are typical.
- Polyuria and polydipsia are late signs, due to renal failure.
- Muscle weakness or tremors may occur acutely.

See main reference on page 245 (The Cat With Polyuria and Polydipsia) for details.

## Clinical signs

**Vomiting** may be due to the disease causing the increased serum calcium or to the direct effects of elevated calcium stimulating vomiting receptors both peripherally or centrally.

**The signs are dependent upon the cause of hypercalcemia**, which can be due to the presence of malignancy, ingestion of cholecalciferol-containing rodenticides, endocrinopathies (primarily primary hyperparathyroidism) or idiopathic.

Signs associated with large elevations in serum calcium include **anorexia, vomiting, muscle weakness, polyuria and polydipsia, shaking/tremors and weight loss**.

Hypercalcemia has also been associated with the **development of calcium-containing nephroliths or uroliths**, which may cause signs of renal or bladder dysfunction.

Generally, **in cats, hypercalcemia is rare**, but if present is most commonly caused by renal failure or is secondary to malignancy. In some cats, the cause cannot

be identified. Other rarer causes include vitamin D toxicosis, primary hyperparathyroidism, hyperthyroidism, hypoadrenocorticoism and granulomatous disease.

## Diagnosis

**Hypercalcemia of malignancy** is most often secondary to lymphoma, but may be due to multiple myeloma, adenocarcinoma and squamous cell carcinoma. Diagnosis of this disorder is based upon finding the primary tumor, an elevation of PTH-rP but not of iPTH in serum, and a response to therapy.

**Primary hyperparathyroidism is rare in cats**, but will be associated with an increased iPTH assay, a decrease in serum phosphorus and lack of other primary abnormalities.

**Cholecalciferol poisoning** is associated with a decrease in both the iPTH assay and PTH-rP assay, hyperphosphatemia and often evidence of mineralization of abdominal and other soft tissues.

**Chronic renal failure (renal secondary hyperparathyroidism)** is associated with an elevated iPTH and PTH-rP assay, but normal or decreased ionized calcium. Ionized calcium will be elevated with all other causes of hypercalcemia.

**Recently, idiopathic hypercalcemia** has been reported in cats and acidifying diets were implicated. This is associated with an increased serum total calcium or serum ionized calcium concentration, or both. Serum iPTH concentrations are low or in the low-normal range, while serum PTHrP is usually undetectable. Serum 25-hydroxycholecalciferol (vitamin D3) concentrations are normal in affected cats, as are serum phosphorus and albumin levels.

**Phosphorous concentrations** are normal or low-normal in all causes of hypercalcemia except cholecalciferol toxicity or renal failure, unless renal failure develops secondary to hypercalcemia.

## Treatment

Specific treatment of hypercalcemia is directed at correcting the underlying cause if possible.

**Supportive therapy** measures to control or reduce hypercalcemia include **correction of fluid deficits and**

**fluid diuresis** with physiologic saline (60–180 ml/kg/day) and **administration of drugs that induce calciuresis** (furosemide 2–4 mg/kg PO q 12 h, or prednisolone 1–2 mg/kg PO q 12 h).

**In severe, life-threatening hypercalcemia**, where the above measures are inadequate, **sodium bicarbonate** (0.5–2 mEq/kg in IV fluids over 6 hours), **salmon calcitonin** (4 U/kg IV q 12–24 h) and **dialysis** (peritoneal or hemodialysis) **may be necessary**.

Other long-term therapeutic measures include feeding **a low-calcium diet** (Hill's k/d, u/d).

## FELINE VIRAL DISEASES (FeLV, FIV, FIP)

### Classical signs

- Weight loss, anorexia and lethargy are the most common signs.
- Vomiting is less common.
- ± Fever

See main reference on pages 540, 339, 372 for details.

### Clinical signs

**Specific signs depend upon the effect of the infection on the individual cat**, e.g. development of neoplastic disease, bone marrow involvement, secondary infections and local (granuloma formation in FIP) or systemic disease (CNS, respiratory, hepatic, effusions).

The **most common signs** are **weight loss, anorexia and lethargy or depression**.

**Feline leukemia virus (FeLV)** is often associated with **neoplastic disease** (lymphoma, bone marrow infiltration) or **bone marrow dyscrasias** (cytopenias, myelodysplasias, etc.). Vomiting may occur associated with lymphoma.

**Feline immunodeficiency virus (FIV)** results in immunodeficiency disease that often results in **secondary infections** being the cause of significant morbidity and mortality. The skin, respiratory system and the gastrointestinal tract are most often affected.

The **classical presentation of effusive feline infections peritonitis (FIP)** is anorexia, lethargy, fever, weight loss, abdominal enlargement and dyspnea due to the **pleural and peritoneal effusion** of fluid.

**Non-effusive FIP** may present with a **wide variety of clinical signs depending on the system involved**, including chorioretinitis, fever, vomiting and/or diarrhea, abdominal pain (renal, liver), anorexia, lethargy and weight loss.

## Diagnosis

**Diagnosis of FeLV** is by **ELISA antigen testing** and confirmation with serum or bone marrow IFA if the diagnosis is in doubt. PCR testing can also be performed in FeLV-suspect cats for confirmation.

**FIV infection** is detected by an **antibody test** and confirmed either via western blot analysis or **PCR testing** for the presence of viral proteins. (Note: vaccinated cats will test positive on the antibody test.)

**Diagnosis of FIP is a very difficult process** because of the lack of a definitive test that is sensitive and specific enough to differentiate enteric corona viral infections from FIP (a mutant of enteric corona virus).

**Antibody titers are available, but detect the presence of any corona virus** (feline enteric, canine enteric, FIP, etc.). Most cats from catteries, shelter situations or large, multi-cat populations will be exposed to enteric corona virus because of the ubiquitous nature of the virus in these settings (up to 85–90%). Thus, antibody testing of cats from these environments will be quite difficult to interpret. **Specific ELISA tests for the 7B protein in FIP have not proved to be reliable in the field.**

**Serum PCR testing** for FIP shows great promise for becoming a definitive diagnostic test, but the currently available serum tests still are not reliable tests for distinguishing FIP versus enteric corona virus. **The PCR test is an excellent means of ruling out FIP (negative test means the cat does not have FIP).** However, **a positive serum PCR test does not prove the cat has FIP.**

The **definitive diagnosis of FIP** still requires **histologic examination of tissues** for the classical changes or virus isolation. This is not usually necessary in cats with effusive FIP, but non-effusive FIP has such variable clinical signs and progression that the diagnosis is quite challenging.

**PCR on tissue samples** which have classic changes is also a good diagnostic test for FIP.

## HISTOPLASMOSIS\*

### Classical signs

- Dyspnea, wheezing or coughing are classical signs.
- Signs of gastrointestinal, CNS, or hemolymphatic system disturbances occur with systemic involvement.

See main reference on page 755 for details.

### Clinical signs

The **classical presenting signs are respiratory: dyspnea, wheezing or coughing.**

In cats with systemic histoplasmosis, the gastrointestinal tract, CNS or the skeletal system may be involved. The **GI tract signs are typically associated with diarrhea**, but in some cases, vomiting is observed, along with anorexia, weight loss and lethargy.

In generalized histoplasmosis, more non-specific signs may occur, such as anorexia, weight loss, fever, lymphadenopathy and lethargy.

Nervous system signs are rare and typically associated with seizures, changes in mentation or meningitis (neck or back pain, other neurologic signs such as ataxia, cranial nerve signs or weakness).

The **hemolymphatic system** involvement is manifest primarily as **bone marrow infiltration** with the fungus, which crowds out the red cell, white cell and/or platelet precursors, so the clinical signs are variable depending on the extent of marrow destruction.

### Diagnosis

This disease has a **regional occurrence**, so a supportive history in a cat from an endemic area is suggestive.

In cats with **pulmonary histoplasmosis**, there may be very few hematologic or serum biochemical changes (e.g. leukocytosis, mild anemia, hyperproteinemia due to increased gamma globulins).

**Disseminated histoplasmosis** will often be associated with increases in liver enzyme activities, abnormal hemogram (bone marrow involvement causing anemia, thrombocytopenia, etc.), or changes associated with

protein losing enteropathy (hypoproteinemia, hypoalbuminemia, hypocholesterolemia, altered serum electrolytes).

**Radiographs** will be useful in cats with pulmonary disease, but less helpful with evaluation of other organs. Cats with intestinal histoplasmosis often have thickened loops of bowel that can be seen radiographically or via ultrasound imaging. **Ultrasound** may be useful in assessing liver and GI tract involvement.

**Serology** is available for detection of systemic fungal infections, including histoplasmosis, but the tests have a **high percentage of false negatives** in cats.

The **definitive diagnosis is obtained by finding organisms** in the affected tissues.

Endoscopic or surgical biopsy, fine-needle aspirates by ultrasound guidance, bone marrow examination or bronchial washing are necessary to obtain the diagnosis. In cats with signs of GI disease, especially diarrhea, rectal scrapings may also reveal the organism.

## TOXOPLASMOSIS\*

### Classical signs

- Toxoplasmosis is a systemic disease involving multiple organs systems.
- Respiratory tract signs (dyspnea, coughing, rhinitis).
- CNS signs (ataxia, behavioral changes, seizures, circling).
- Hepatic disease signs (vomiting, diarrhea, icterus, anorexia, lethargy, weight loss, abdominal effusion, or discomfort).
- Cardiac signs (arrhythmias, sudden death).
- Ocular signs (chorioretinitis, anterior uveitis, optic neuritis, blindness, anisocoria, glaucoma or retinal detachment).
- Rarely GIT granuloma causing vomiting.

See main reference on page 375 for details.

### Clinical signs

**Toxoplasmosis is a systemic, multi-organ disease that often affects young kittens**, but adult cats are also

infected, especially those with FIV or feline leukemia virus infection.

**Infected cats most frequently have respiratory tract disease** (dyspnea, coughing, rhinitis), **CNS disease** (ataxia, behavioral changes, seizures, circling), **hepatic disease** (vomiting, diarrhea, icterus, anorexia, lethargy, weight loss, abdominal effusion or discomfort), **cardiac disease** (arrhythmias, sudden death) or **ocular disease** (chorioretinitis, anterior uveitis, optic neuritis, blindness, anisocoria, glaucoma or retinal detachment).

Adult cats with FIV have more multi-systemic signs, while cats without FIV often have ocular or neurologic signs only.

Occasionally, toxoplasma granulomas (tissue cysts) form in the GIT or pancreas, rather than encysting in muscle, and result in chronic vomiting. The encysted organisms cause immune complex formation that is responsible for the granuloma formation and the chronic, but sublethal clinical disease.

### Diagnosis

**Hemogram abnormalities: non-regenerative anemia, neutrophilic leukocytosis, lymphocytosis and eosinophilia** are most commonly observed.

Lymphopenia may be present in cats with FIV or end-stage disease.

**Biochemical abnormalities** are common with systemic disease and include: hypoproteinemia, hypoalbuminemia, hyperglobulinemia, elevated liver enzyme activities (hepatic involvement), increased creatine kinase (muscle involvement), hyperbilirubinemia (cats with cholangitis), proteinuria is also relatively common.

In focal GIT *Toxoplasma* granuloma, biochemical abnormalities may be minimal.

**Diagnosis is based on finding the organism in tissues (cytology or histopathology) or by serologic testing.**

**Thoracic radiographs** in cats with acute disease will reveal a diffuse interstitial pattern with a mottled lobar distribution.

**Fecal examination for oocysts in cats is usually not helpful** since less than 1% of infected cats shed the oocysts on any given day.

**Serologic testing** is currently the best method of detection, but because of the presence of antibodies in both healthy and diseased cats, titers must be used cautiously. **No single serologic test can definitively confirm toxoplasmosis.**

Serum ELISA tests for IgM, IgG and IgA are readily available and have been used to try to distinguish acute from chronic infections in cats. Finding of a **high IgM titer, along with a four-fold increase over 2–3 weeks (or decrease) in IgG titer** (in the presence of appropriate clinical signs, and response to anti-toxoplasma drugs, e.g. clindamycin) is suggestive of recent or active toxoplasmosis.

Other tests of serum IgG supportive of toxoplasma infection are the **modified agglutination test (MAT)** and the latex agglutination test (LAT). The MAT is the most sensitive test of IgG antibody, but is not uniformly available. The LAT cannot distinguish antibody classes, thus is less useful.

Aqueous humor and CSF titers are also useful, but should be compared with serum titers to determine if local immunoglobulin production is occurring.

## INTESTINAL MAST CELL TUMOR\*

### Classical signs

- Weight loss and anorexia are the most common early signs.
- Intermittent or persistent vomiting, diarrhea and lethargy or depression become more prevalent with advancing disease.

### Pathogenesis

Mast cell tumors of the intestine are the **third most common GI tract neoplasm** in cats behind lymphoma and intestinal adenocarcinoma.

These are tumors of **old (mean age = 13 years) cats**, with **no breed or sex predilection**. They occur with about the same frequency as mast cell tumors of the lymphoreticular system (e.g. spleen, lymph nodes and bone marrow), but **cats with intestinal mast cell tumors typically do not have circulating mast cells.**

**Intestinal mast cell tumors are usually solitary**, and affect the **distal small intestine**, but multiple masses can occur. Typically, they appear as a **segmented nodular thickening in the small intestine.**

Mast cell tumors of the GI tract are usually **poorly differentiated** (thus cytologic examination is difficult) and **highly malignant**, with metastasis to the mesenteric lymph nodes, liver and spleen. They are generally not functional tumors and are **not typically associated with gastrointestinal ulcer disease**. However, there may be chronic bleeding from the tumor into the GIT.

**No association with FeLV, FIV or FIP** has been reported.

### Clinical signs

The **most common clinical signs** in cats with an intestinal mast cell tumor are **weight loss, lethargy and anorexia.**

Intermittent or persistent **vomiting, diarrhea and lethargy** or depression become **more prevalent with advancing disease.**

Vomiting and/or diarrhea may also be observed, but often not until late in the disease.

Other signs include ascites, splenic enlargement (metastasis), and rarely, mast cells in circulation (bone marrow involvement).

### Diagnosis

**Palpation of an intestinal mass** in an older cat is suggestive of neoplasia, but **these tumors may be very small.**

**Abnormalities of the hemogram or serum chemistry profile are uncommon**, but may include anemia (non-regenerative), hypoproteinemia and elevated liver enzyme activities.

**Survey radiographs** may identify a mass, evidence of intestinal obstruction, or mild peritoneal effusion, but contrast radiography will be required to identify the location and extent of many of these tumors.

**Ultrasonography** is a useful tool in identifying the location, extent and presence of metastatic lesions. **Fine-needle aspiration or biopsy** of these masses by

ultrasound guidance may be useful, but because of the poorly differentiated nature of the tumor they may also be non-diagnostic.

**Analysis of peritoneal effusion fluid** may be helpful if mast cells are present, but as with fine-needle aspirates, the effusion may be suggestive of neoplasia, but not necessarily give a definitive diagnosis.

**Definitive diagnosis requires histopathologic examination** of the tissue, which is usually best achieved by wide surgical resection and anastomosis.

**Staging of the tumor** is achieved by biopsy of mesenteric lymph nodes, spleen, liver and bone marrow aspiration (if indicated). **The tumor rarely metastasizes out of the abdomen.**

### Differential diagnosis

Other intestinal neoplasia, foreign body, fungal or algal disease, inflammatory bowel disease, and extra-intestinal causes of anorexia, weight loss and vomiting (chronic renal failure, hyperthyroidism, etc.) are all differentials that should be considered.

### Treatment

**Surgical resection is the only known treatment.** Surgical margins must be > 5 cm beyond the visible edge of the lesion because of the cellular invasion that occurs along the tumor mass.

**Metastasis occurs early in the course of the disease** to the spleen, liver and regional lymph nodes.

Other forms of therapy (**chemotherapy, radiation, etc.**) **have either been ineffective or not evaluated.** **Adjunct therapy with cimetidine, anti-histamines and prednisone** may be tried to control signs associated with mast cell degranulation, but are **often not helpful** because these tumors are typically poorly differentiated and **do not produce granules or GI ulcer disease.**

**Nutritional support** (enteral or parenteral nutrition) may be required for cats that do not want to eat or are vomiting.

### Prognosis

The **prognosis is poor** for cats with this disease.

Despite surgical removal, **cats with intestinal mast cell tumors rarely live longer than 4 months.**

## FOREIGN BODIES\*

### Classical signs

- The classical sign is an acute onset of frequent vomiting.
- Intermittent vomiting will occur in some cats with foreign bodies that are causing an intermittent or incomplete obstruction (e.g. string).

See main reference on page 636 for details.

### Clinical signs

The **classical sign is an acute onset of frequent vomiting**, but intermittent vomiting will occur in some cats with foreign bodies that are causing an **intermittent or incomplete obstruction** (e.g. string).

There may or may not be palpable abnormalities in the intestinal tract, and most cats will be clinically normal otherwise.

### Diagnosis

The **definitive diagnosis** is usually made by **contrast radiography**, however, survey radiographs or **ultrasound examination** may also reveal abnormalities that are consistent with a GI foreign body.

String foreign bodies often are found attached to the base of the tongue, thus a **thorough oral exam is always essential in cats with a suspected GI foreign body.**

Some foreign bodies can be found and retrieved by **gastrointestinal or colonic endoscopy**. In many cases, these foreign objects are metallic and will be visible on survey radiographs.

### Differential diagnosis

Gastric parasites, heartworm disease, *Helicobacter* spp. gastritis, food intolerance, food allergy, and antral pyloric hypertrophy or stenosis are primary differentials

because they all may cause vomiting in the absence of other signs of disease.

## Treatment

**Surgical removal of the foreign body** is required if it is not able to be retrieved and removed via an endoscopic procedure.

## CONGESTIVE HEART FAILURE\*

### Classical signs

- Coughing or dyspnea.
- Lethargy.
- Anorexia.
- Exercise intolerance.
- Weight loss.

See main reference on page 124 for details (The Cat With Abnormal Heart Sounds and/or an Enlarged Heart).

## Clinical signs

**Classical signs** of congestive heart failure include **dyspnea, coughing, cyanosis, pale mucous membranes**, poor pulse quality, inappetence, exercise intolerance/weakness and lethargy. Cats are usually presented with acute dyspnea and cyanosis.

The **signs may be quite variable** depending on the cause of heart failure. **Valvular disease** is often associated with an **audible murmur**, while **heart muscle diseases** often are associated with **tachycardia, gallop rhythm and weak pulses**.

Other signs that may be observed in cats with heart failure include collapse, vomiting, weight loss, limb paresis/paralysis due to thromboembolic disease, and rarely, abdominal distention.

Other cats may develop pleural effusion that increases respiratory effort but is not associated with coughing or increased lung sounds (decreased or absent lung sounds are present).

**Vomiting episodes are typically chronic, but infrequent** and more commonly associated with heart muscle disease (myopathy) rather than valvular disease.

## Diagnosis

**History and physical examination findings will be suggestive** of congestive heart failure, especially if the cat has a murmur, gallop rhythm or other signs referable to primary heart disease.

**Hematology and serum biochemical profile data** may reveal **mild, non-specific changes**, such as anemia of chronic disease, prerenal azotemia, elevations in liver enzyme activities secondary to congestion, and changes in electrolytes and proteins consistent with dehydration. If myositis is a differential for the cause of heart failure, elevated creatine kinase values may be useful additional information.

**Imaging studies** are necessary to make a **definitive diagnosis**. In particular, **thoracic radiographs** will be helpful in assessing **cardiac size and pulmonary involvement** (edema vs. pleural effusion, etc.). **Cardiac ultrasound examination** is the best diagnostic test for evaluating cardiac muscle function, size and presence of valvular or other defects.

**Electrocardiography** may also be useful to detect or document any **arrhythmias**.

## SYSTEMIC NEOPLASIA\*(GENERALIZED LYMPHOMA, SYSTEMIC MASTOCYTOSIS)

### Classical signs

- Weight loss.
- Lethargy or depression.
- Anorexia.
- Enlarged spleen or liver, lymph nodes or bone marrow involvement.

## Clinical signs

The **most common clinical finding** is marked **hepatomegaly or splenomegaly** along with **enlarged lymph nodes** (mesenteric).

Other common signs are **vomiting and anorexia**.

In some cases, **peritoneal effusion** will be present, which along with the hepatomegaly or splenomegaly will be observed as **profound abdominal enlargement**.

## Diagnosis

**Definitive diagnosis** is obtained by **fine-needle aspiration of the enlarged liver, spleen or lymph nodes**.

Mast cells or lymphoblasts/cytes may also be found in the systemic circulation on routine hematology, and will also be found on examination of **bone marrow aspirates**. In addition, **anemia and cytopenias are also common findings** on hematologic exams.

**Ultrasound or radiographic evaluation** will reveal the extreme size of the liver and spleen and ultrasound guidance can be used in obtaining fine-needle aspirates (especially of lymph nodes or sections of the liver that have obvious structural abnormalities).

## CNS DISEASES\* (ENCEPHALITIS, VESTIBULAR DISEASE, SEIZURE DISORDERS, NEOPLASIA)

### Classical signs

- Neurologic signs localized to the region of the CNS that is affected (e.g. vestibular disease is associated with loss of balance, ataxia, falling to one side, head tilt, nystagmus, etc.).
- Vomiting is infrequent.

## Clinical signs

The predominant clinical signs will be **neurologic signs that are localized to the region of the CNS that is affected**. Vestibular disease can be associated with vomiting, but typical signs include loss of balance, ataxia, falling to one side, head tilt and nystagmus. **Most neurologic diseases that cause vomiting affect the CNS**, not the spinal cord or peripheral nervous system, except for the peripheral parts of cranial nerve VIII located in the inner ear.

**Vomiting is an infrequent but still important sign of CNS disturbance** most often associated with involvement of the **peripheral vestibular system**.

Cats with signs of **hepatoencephalopathy** (changes in behavior, stupor, drooling or ataxia) may vomit from the effects on the CNS or the GIT.

## Diagnosis

A thorough **neurologic examination** is the first step in determining if neurologic disease is present and in attempting to determine if the disease is affecting the central, spinal or peripheral nervous system.

**Other important diagnostic tests for diseases in the CNS** include CSF analysis, including culture and serology (FIP, toxoplasmosis, fungal disease), computed tomography or MRI imaging, electroencephalogram, and brainstem auditory-evoked response (middle/inner ear).

**Radiographs of the middle/inner ear areas are non-specific** and very difficult to evaluate, and have **largely been replaced by CT** as a means of assessing this region. However, **middle and inner ear infections** can also be diagnosed via **myringotomy and culture of fluid** from the middle ear.

Unless a systemic disease process is the cause of the CNS signs, the **minimum data base is likely to be normal**.

## PHYSALOPTERA

### Classical signs

- The classical sign is frequent vomiting, but intermittent vomiting will occur in some cats.

See main reference on page 653 for details (The Cat With Signs of Acute Vomiting).

## Clinical signs

The **predominant clinical sign is vomiting**. Most cats will present with an acute onset of vomiting, but others may have intermittent, chronic vomiting.

## Diagnosis

**Fecal floatation** and finding of the **larvated eggs** in the feces (**shedding is intermittent**, so multiple feces are required) is diagnostic, but unrewarding.

Finding of **small worms in vomitus or on endoscopic examination**.

Response to a **therapeutic trial with fenbendazole** (25 mg/kg PO q 24 h for 3–5 days).

## HEARTWORM DISEASE (*DIROFILARIA IMMITIS*)

### Classical signs

- Signs are variable.
- No signs may be present.
- Signs of respiratory disease (tachypnea, dyspnea).
- Intermittent vomiting.
- Signs of cardiovascular disease (lethargy, anorexia, coughing, weakness).
- Sudden death.

See main reference on page 139 for details.

### Clinical signs

**Anorexia and weight loss** are the **most common signs** observed in cats with dirofilariasis.

Respiratory signs such as wheezing, dyspnea, and occasionally coughing are less common than in dogs, and often **mimic feline asthma**.

**Intermittent vomiting** is a relatively frequent sign associated with feline heartworm disease that may occur in the absence of other clinical signs.

Sudden death is a complication of heartworm disease in cats, and may occur in 20–30% of infected cats.

### Diagnosis

A history of **living in a heartworm endemic area** along with the **appropriate clinical signs** in the absence of heartworm preventative use should arouse clinical suspicion.

Hematology may reveal an **eosinophilia or basophilia**.

Unless cats have developed heart failure, the chemistry profile and urinalysis are usually normal. Cats that are in right congestive heart failure may have elevated liver enzyme activities or azotemia.

Radiographs of the thorax will reveal the **classic changes associated with heartworm disease**, which are enlarged pulmonary arteries, arterial tortuosity or blunting, pulmonary interstitial pattern and right-sided cardiac enlargement.

**Ultrasound** can also be used to identify worms in the main pulmonary artery or right ventricle, but the sensitivity of this test is highly dependent on operator experience.

A **heartworm antibody test** is the recommended **screening test** for cats, and while it does not confirm infection, it is an accurate means of confirming exposure. However, **it may remain positive for 18 months or more after resolution of infection, and also may be positive if arrested larval infection has occurred**.

Because cats generally have only 1–2 worms present, the **standard heartworm antigen tests used in dogs are often not sensitive enough to detect the disease in cats**.

A feline antigen test has been developed, and detects the presence of a single male worm, but false-negative tests still occur. Antigen tests should not be the sole means of evaluating cats for heartworm infection.

## HYPOADRENOCORTICISM (ADDISON'S DISEASE)

### Classical signs

- Lethargy, anorexia and weight loss are most common.
- Vomiting.
- Polyuria, polydipsia.
- Clinical signs may wax and wane.

See main reference on page 252 for details.

### Clinical signs

**This is a very rare disease in cats.**

The classical signs are **lethargy, anorexia and weight loss**. Other signs that may be seen are vomiting, polyuria/polydipsia, weakness and waxing/waning signs.

Most cats (> 50%) will be **hypothermic, dehydrated or depressed on physical examination**, but other signs such as weak pulses, bradycardia and collapse are less common.

### Diagnosis

The **history and clinical signs will be vague in early cases**, so that a degree of clinical suspicion is required to make the diagnosis.

**Anemia, lymphocytosis and eosinophilia are relatively uncommon (< 20%) findings in cats.**

Serum biochemistries and urinalysis show **hyponatremia, azotemia (pre-renal or renal), and hyperphosphatemia** in the majority of cats. Hyperkalemia and hypochloridemia are also common, but not seen in every case.

Other less common (< 30% cases reported) abnormalities include metabolic acidosis, increased liver enzyme activities, hypercalcemia and hyperbilirubinemia.

Slightly more than 50% of affected cats will have an **unconcentrated urine sample (SG < 1.030)**, supporting renal azotemia.

The **definitive diagnostic test** for hypoadrenocorticism is an **ACTH stimulation test**, which will reveal a low resting cortisol concentration, and no response (no increase in cortisol levels) following administration of ACTH (Cosyntropin 0.125 mg/cat, IM), at either the 30 or 60 min post-administration sampling times. Administration of steroids by any route (oral, parenteral or topically) or progestins (e.g. megestrol acetate) will suppress cortisol response to ACTH. This suppression may persist for weeks after the last time of administration, especially if administration was chronic.

**Radiographic changes** include microcardia and hypoperfusion of the lungs due to hypovolemia. **ECG changes classically observed in dogs**, such as peaked T waves, reduced or absent P waves, or atrial standstill are not seen in cats.

If serum electrolyte concentrations are normal, but a subnormal response to ACTH administration is observed, the cat may have: (1) residual mineralocorticoid secretion; (2) secondary hypoadrenocorticism from pituitary or hypothalamic disease causing only glucocorticoid deficiency but this has not yet been reported in cats; or (3) **secondary hypoadrenocorticism due to exogenous administration of glucocorticoids or progestogens**. Although this is the most common cause of secondary hypoadrenocorticism and suppressed adrenal response to ACTH, it rarely causes sufficient lethargy for veterinary attention to be sought. If clinical signs consistent with hypoadrenocorticism are present in a cat with a suppressed ACTH stimulation test secondary to steroid administration, the signs are most likely caused by another disease.

To differentiate pituitary or hypothalamic disease from exogenous drug administration as the cause of secondary hypoadrenocorticism, **plasma ACTH concentration** is measured.

## INTUSSUSCEPTION

### Classical signs

- The classical sign is an acute onset of frequent vomiting.
- Intermittent vomiting will occur in some cats.

See main reference on page 643 for details.

### Clinical signs

An **acute onset of vomiting, abdominal pain or anorexia is the most common sign**. However, in some cats with **sliding, intermittent or incomplete intussusception**, the signs may be intermittent. **Weight loss and vomiting** will be more prevalent in these cats.

### Diagnosis

A palpable abdominal mass may help direct the diagnostic approach if it is present.

**Contrast radiographs or ultrasound examination** of the GI tract are the most definitive means of making the diagnosis.

**Intussusception rarely presents as a chronic problem**, so in these cases, abnormalities associated with chronic GI dysfunction (e.g. hypoproteinemia, hypoalbuminemia) may be present.

## LEAD POISONING

### Classical signs

- Gastrointestinal signs include anorexia, vomiting, diarrhea and abdominal pain.
- Neurologic signs include aggression, nervousness, tremors, seizures, blindness and dementia.

See main reference on page 596 for details.

## Clinical signs

**Gastrointestinal signs** include anorexia, vomiting, diarrhea and abdominal pain. Weight loss is common.

**Neurologic signs** include aggression, nervousness, tremors, seizures, blindness and dementia.

**GI signs** tend to occur **acutely** and **before neurologic signs**. **CNS signs** are associated with **severe toxicity or chronicity**.

## Diagnosis

There may be a history of exposure to lead-containing materials in the environment (paints, roofing materials, batteries, used motor oil, lead shot, etc.).

Characteristic clinical signs (the **combination of gastrointestinal and neurologic signs**) are suggestive.

There are **typical hemogram changes** including basophilic stippling of RBCs, and increased numbers of nucleated RBCs, although these are not as common in the cat as in the dog.

**Radiographs** may reveal radiopaque material in the GIT.

The **definitive diagnosis** is made by measurement of a **blood lead level** (> 0.5 ppm is diagnostic).

## Treatment

**Gastric lavage** or **induction of emesis** should be performed to remove the offending materials from the stomach, **if ingestion was recent**. If the material can be retrieved by endoscopy or surgical removal (e.g. it is a large object) it should be done.

**Oral activated charcoal** should be administered to reduce the absorption of the material from the GIT.

An IV catheter should be placed and **supportive fluid therapy** (Normosol or LRS, 40–60 ml/kg IV) should be commenced.

**Calcium EDTA** to chelate lead and hasten excretion can also be used (25 mg/kg q 6 h IV as 10 mg Ca EDTA/ml in dextrose, maximum 2–5 days).

If seizures or other severe neurologic signs are present, **appropriate seizure control** (e.g. IV valium 2.5–5 mg or phenobarbital 1–4 mg/kg IM) should be used.

If the cat is debilitated and anorectic, **nutritional support** may also have to be instituted.

## OTHER INTESTINAL NEOPLASIA (FIBROSARCOMA, CARCINOMAS, PLASMOCYTOMA, LEIOMYOSARCOMA, ETC.)

### Classical signs

- Weight loss.
- Lethargy or depression.
- Anorexia.
- Vomiting and/or diarrhea.

## Clinical signs

The **clinical signs observed** will depend upon the **tumor location** in the GI tract.

In cats, **anorexia, weight loss and lethargy are the most common clinical signs** of any gastrointestinal neoplastic disease.

**Vomiting and/or diarrhea may be observed, but will be variable** depending on whether the tumor is in the small intestine or colon, and whether it is diffuse or solitary in extent.

## Diagnosis

**Palpation of an intestinal mass is an important diagnostic tool**, since many of these tumors are solitary and produce strictures or obstructions.

**Hematology and serum biochemistry profiles** may be **within normal limits** in cats with intestinal neoplasia.

Abnormalities associated with secondary effects of the tumor include dehydration, electrolyte imbalances, anemia of chronic disease, etc.

**Radiographs** of both the thorax and abdomen are an important, but sometimes non-diagnostic, tool. Radiographs are best for assessment of **pulmonary metastasis**.

Ultrasound is an invaluable tool for lesion localization, determination of lesion extent and in some cases, obtaining a fine-needle aspirate or biopsy of the lesion.

Definitive diagnosis is by histopathologic evaluation of a biopsy or tissue obtained after surgical resection.

## ANTRAL PYLORIC HYPERTROPHY/STENOSIS

### Classical signs

- Vomiting several hours after eating.
- Projectile vomiting will be observed if the pylorus is obstructed.
- Abdominal distention or pain may be observed due to gastric distention.

### Pathogenesis

Antral pyloric hypertrophy is a condition that involves hyperplasia of the pyloric musculature, pyloric mucosa or both, which results in narrowing or complete obstruction of the gastric outflow tract.

The condition can be congenital or acquired, but in either case, the exact cause is unknown.

Both neural dysfunction and endocrinopathies (hypergastrinemia) have been proposed to be involved in the disease.

The obstruction of gastric outflow results in gastric retention and subsequent gastric distention. Gastric distention stimulates gastrin secretion which is trophic to antral tissues and may further contribute to mucosal hypertrophy.

In some cases of congenital pyloric stenosis there is no antral hypertrophy. The gastric retention is believed to be due to a motility disturbance that results in a functional obstruction.

For reasons that are yet unknown, the disorder has been observed primarily in Siamese cats.

### Clinical signs

Vomiting is the most common sign.

In congenital pyloric stenosis, kittens begin to vomit shortly after they commence solid food.

Acquired pyloric hypertrophy has a more variable presentation, but is usually associated with intermittent vomiting of digested food several hours after consumption. If the pylorus is completely obstructed the vomiting may be projectile.

Chronic intermittent vomiting and gastric distention may lead to gastroesophageal reflux or esophagitis which may be associated with regurgitation or inappetence and weight loss.

### Diagnosis

History and signalment will be suggestive of a gastric emptying disturbance.

Hemogram, serum chemistries and urinalysis are typically unremarkable.

Survey radiographs of the abdomen may be normal, show gastric distention, or pyloric abnormalities. Contrast radiographs may identify hypertrophic gastric mucosa or a narrow gastric outflow pathway ("beak sign").

Ultrasonography may detect a thickened pyloric antrum if it is present.

Fluoroscopy or scintigraphy can be used to evaluate gastric peristalsis and emptying, but are not universally available.

Gastric endoscopy may be normal or may reveal thickened folds of mucosa, the pyloric antrum may not insufflate normally, or the pylorus may be too stenotic to allow passage of the endoscope through the orifice.

Definitive diagnosis requires an exploratory laparotomy and histopathologic examination of tissue for antral pyloric hypertrophy, but motility disturbances can only be diagnosed via fluoroscopy/scintigraphy, and hypergastrinemia can be detected by measurement of serum gastrin levels.

### Differential diagnosis

Neoplasia (gastric adenocarcinoma, leiomyosarcoma, lymphosarcoma) of the gastric, pancreatic or duodenal tissues resulting in pyloric thickening and obstruction should be considered, especially in older cats.

Neoplasia of surrounding tissues resulting in gastric outflow obstruction from an external source, or a gastric

foreign body resulting in obstruction or decreased outflow may also result in similar clinical signs.

Gastric ulcer disease may result in chronic vomiting and abnormal motility.

Extra-gastrointestinal causes of vomiting include hepatic, renal, metabolic, inflammatory (pancreatitis) or endocrine causes.

## Treatment

Definitive treatment for pyloric antral hypertrophy/stenosis is **surgical correction of the outflow obstruction**.

In cats with **motility disturbances**, **gastric prokinetic therapy** (metoclopramide 0.1–0.2 mg/kg PO q 12 h, or cisapride 5 mg/cat PO q 8–12 h) may improve movement of ingesta and reduce clinical signs.

However, **in early cases**, medical management with **feeding of small meals consisting of highly digestible foods** (low residue, low fat, low fiber) may also be helpful.

Following surgical correction of the outflow obstruction, some animals will still require medical therapy with gastric prokinetics because of residual motility disturbances.

## Prognosis

The prognosis is **guarded to good with surgical management**, as some animals may still require life-long medical management following surgery to control the clinical signs.

Cats that develop **esophagitis due to reflux disease** may have a **more guarded prognosis** because of the problems associated with esophagitis (stricture development).

## DYSAUTONOMIA

### Classical signs

- Depression and anorexia.
- Mydriasis, reduced pupillary light reflex, protruding nictitans, normal vision.
- Xerostomia (dry mouth and mucous membranes) and keratoconjunctivitis sicca.
- Retching, regurgitation or vomiting.
- Fecal incontinence or constipation.

See main reference on page 792 for details (The Constipated or Straining Cat).

## Clinical signs

**Lethargy and anorexia are present in almost all cats** and usually develop over a **48-hour period**.

The classical signs are associated with **the dysfunction of both sympathetic and parasympathetic nervous system**. **Ocular signs include** dilated pupils, decreased or absent pupillary light reflex and protruding nictitans, but have **normal vision**.

**Gastrointestinal signs are present in about 95% of cats** and include constipation, regurgitation (secondary to megaesophagus) and dry mouth. Vomiting may occur or regurgitation may be reported as vomiting.

Less commonly observed signs are bradycardia, hypotension, urinary incontinence or a distended, easily compressed bladder, proprioceptive deficits and transient syncopal episodes.

## Diagnosis

There is **no definitive, antemortem diagnostic test for dysautonomia**.

Diagnosis is made by using the **clinical presentation** along with **radiographic evidence of megaesophagus** and its associated complications, and use of **pharmacologic testing** to evaluate the function of the sympathetic and parasympathetic nervous system.

**Direct-acting parasympathomimetic and sympathomimetics produce an effect in diseased cats** but not in normal cats because of post-ganglionic denervation hypersensitivity in affected cats. **Indirect (preganglionic) acting agents produce an ocular effect in normal cats but not diseased cats**.

Ocular testing with sympathomimetic agents, e.g. 1% hydroxyamphetamine (indirect acting) produces no mydriasis, and 1% phenylephrine (direct acting) produces rapid mydriasis in affected cats.

Ocular testing with parasympathomimetic agents, e.g. 0.5% physostigmine (indirect acting) produces no miosis, and 0.1% pilocarpine (direct acting) produces rapid miosis in affected cats.

Systemic testing using 0.04 mg/kg atropine, subcutaneously, will increase the heart rate if the sympathetic nervous system is normal. Note: Do not use ephedrine, epinephrine or other sympathomimetics, because fatal arrhythmias can be induced.

Failure to demonstrate a wheal and flare response following intradermal injection of histamine (1:1000) indicates a defect in the sympathetic innervation of cutaneous blood vessels.

## GASTRINOMA

### Classical signs

- **Chronic vomiting unresponsive to routine therapy for gastritis and gastric ulcer disease.**

### Pathogenesis

**Chronic gastritis and gastric ulcer disease** are produced secondary to a **gastrin-secreting tumor** that arises from the **pancreatic islet cells** (called Zollinger–Ellison syndrome).

This is a **rare disease** that has only been described in three older cats.

The pancreatic tumor may occur as a solitary lesion, or there may be multiple lesions on the liver due to metastasis.

### Clinical signs

Typical signs include **chronic vomiting, weight loss and anorexia** in a cat previously diagnosed with gastritis or gastric ulcers on histopathologic examination of the stomach, but **unresponsive to routine therapy**.

## Diagnosis

The gastritis and gastric ulcer disease are diagnosed by **endoscopy and histologic examination of the stomach and duodenum**.

However, to make a definitive diagnosis of Zollinger–Ellison syndrome, a **serum gastrin level** must be obtained along with the finding of a **discrete tumor on the pancreatic islets**. **Very high levels of serum gastrin** are obtained in cats with gastrinoma.

## Differential diagnosis

These are rare tumors of the GI tract, so **even finding elevated serum gastrin levels is not sufficient**, since there are other causes for elevations in serum gastrin levels (e.g. chronic renal failure, etc.).

Other causes of chronic gastritis, including *Helicobacter* spp., liver disease and pancreatitis should be explored.

## Treatment

Surgical removal of the tumor, if possible, is the treatment of choice. Management of the gastritis and gastric ulcer disease is as for other primary causes of this problem (see section on gastritis for details, page 680).

## RECOMMENDED READING

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